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W. R. BIRDSALL, M.D., L. FISKE BRYSON, M.D., C. L. DANA, M.D., MATTHEW D.
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THE
Journal
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Original Articles.

THREE DIAGNOSTIC SIGNS OF MELANCHOLIA.¹

BY LANDON CARTER GRAY, M.D.,

Professor of Nervous and Mental Disease in the New York Polyclinic.

TRUE melancholia may be divided into simple melancholia, melancholia agitata, melancholia attonita, and melancholia with stupor. It is mainly of the first of these forms, of the slighter cases of the second form and of some few cases of the fourth type that I propose to speak. In the simple form of melancholia, as we all very well know, there is a melancholy, which is usually unattended by delusions, hallucinations or illusions, although there may occasionally be delusional or hallucinatory tendencies, but there is none of the agitation or stupor of the other forms. The simple forms of melancholia are often extremely difficult to diagnosticate, especially in the early stage, as the reasoning powers, the memory, and the perceptions are then often seemingly unimpaired, or not more affected than is possible from a myriad unimportant causes. Patients suffering from this mental disease too frequently figure as neurasthenics, to be confidently treated as such until some determined and frightful suicidal or homicido-suicidal attempt throws startling light upon the true nature of the malady. These, too, are the cases of unaccountable

¹ Read before the American Neurological Association, June 28, 1889, at Long Branch.

suicides which puzzled friends and competing newspaper reporters account for so satisfactorily and sensationally upon some theory of rejected love or high-flown sentimentalism. Any certain diagnostic symptoms in this class of cases should be for these reasons of value. In sixteen cases of melancholia of the above types which I have had under observation in the last eighteen months, I have found three constant symptoms, viz., the melancholia, marked insomnia, and a post-cervical ache.

The melancholy is different from simple sadness. In the latter there is some cause for the mental depression which the patient recognizes and seeks to have removed, and the mental faculties are not at all affected. In melancholia, on the contrary, there is seldom any adequate cause for the depression which the patient will often not recognize the presence of, and about the treatment of which he or she is often utterly indifferent, if indeed, he or she be not obstinately opposed to any treatment; and a careful examination will show a certain dullness of mental reflex, made perceptible by slower responsive acts or words than is usual to a healthy mind. In the more marked cases of simple melancholia, especially in those bordering upon the agitated and stupid types, careful and close questioning will elicit from the patient a history of delusion and hallucinations—rarely illusions, if I may judge by my own experience—of a melancholy and more or less terrifying nature. The characteristics of this peculiar melancholy—the causelessness, the indifference, the slow mental reflexes, with the occasional history of terrifying delusions and hallucinations—are remarkably constant, and they are so significant that I have again and again based upon them a diagnosis which further examination has verified.

The post-cervical ache is, as I have indicated by the name which I have ventured to give it, an aching pain in the back of the neck, head, and sometimes into the shoulders. It is usually described as a distress or ache, although it may occasionally be neuralgic in its character, and not infrequently passes into neuralgic paroxysms which will last for a day or two.

The insomnia is usually one of the earliest symptoms of the disease, but has no especial characteristics of which I am aware.

The following histories will demonstrate that these three conjoined symptoms of melancholia, insomnia and post-cervical ache were constant.

CASE I.—Miss —, age thirty-five, had a severe attack of melancholia three years ago, caused by the sudden death, by a fall from his horse a few days before the intended marriage, of the gentleman to whom she was betrothed. The melancholia lasted for upwards of a year, and it was not until about a year ago that she came under my care. She then complained greatly of obstinate insomnia, and severe aching in the back of the head and neck. For a year this patient has been under my treatment, so that I have been able to observe in her the existence of a fixed melancholy, of such a nature that it seems utterly impossible for her to receive a pleasant impression of any kind. At times she has attacks of dull aching pain in the back part of the head, neck and shoulders, and at the same time she becomes more or less sleepless. These attacks have usually been mild in character, and have yielded after about a week of treatment, but occasionally they have become very severe, and the post-cervical ache has become actual pain of considerable intensity, the insomnia has become pronounced, and the melancholiac condition had deepened and has been attended with mental confusion and hallucinations.

CASE II.—Mrs. B., widow, had attack of melancholia agitata about eight years ago, during which she made repeated and determined attempts at suicide. When I saw her a year or two after the acute symptoms had disappeared she had a fixed melancholy which no reasoning could influence; slept only three or four hours each night, and was troubled with a constant aching, uneasy feeling in the back of the neck and head and in the shoulders. During the years that have since elapsed, in which this patient has either been under my care or so situated that I have known accurately of her condition, she has had almost uninterruptedly the fixed melancholy, the poor sleep and the post-cervical aching. At times the insomnia has become very obstinate, and the post-cervical ache has mounted into severe neuralgic distress, but she has had no increase of her mental symptoms.

CASE III.—Mrs. M., sent to me September, 1888, by a physician. She gave a history of a meningitis following insolation many years ago. During the last six months she has been suffering from melancholia, obstinate insomnia, slight hallucinations, and constant distressing aching in the back of the head and neck and in the shoulders. Under treatment these symptoms disappeared, with the exception of some slight aching in the locality described, and the patient was doing very well when she was again sunstruck in the early part of the summer, and had another attack of meningitis, since when I have lost sight of her.

CASE IV.—Miss —, brought to me by a physician, had had melancholia for some two months, and had made two determined attempts at suicide. She had a causeless melancholy which could not be reasoned away, but had never had any hallucinations or mental confusion, had obstinate insomnia, and well-marked aching in the back of the head and neck and in the shoulders. This patient has improved very greatly during the year that has since elapsed, I am told, but she still suffers from her melancholy, occasional insomnia, but I have not been able to inform myself in regard to the post-cervical aching, as I have never seen her but upon the one occasion of the consultation with her attending physician.

CASE V.—Miss —, age forty, seen in consultation in a neighboring city. She has a causeless melancholy, slight hallucinatory tendencies, has talked longingly of suicide, but has made no attempt at it; has suffered from obstinate insomnia, and has considerable distress in the back of the head. This patient refused to be treated, and I know nothing further accurately of her history.

CASE VI.—Miss —, age thirty-two, seen in consultation. She had had slight melancholy, entirely causeless; had spoken about suicide, but never made any attempt at it; suffered from insomnia for about two weeks, and also from distress in the back of the head and neck. This case had made an excellent recovery in some six weeks under treatment of the attending physician, and when I saw her in consultation she was only suffering from the distress in the back of the head and from slight melancholy. This was altogether one of the mildest cases that I have ever seen in my life.

CASE VII.—Miss —, age twenty-five. This patient had a severe attack of melancholia, with considerable agitation, obstinate insomnia, marked suicidal tendencies, but manifested no symptoms of the post-cervical aching. I attended her for some eight weeks, during which her condition did not materially improve, except that the insomnia was successfully combatted with hypnotics. She then passed out of my charge; whether she afterwards had a post-cervical ache or not, I am unable to say, or as to what her further symptoms were.

CASE VIII.—Miss —, age twenty. This was a case of mild melancholia agitata, with marked melancholia and melancholy hallucinations and some slight mental confusion. This patient committed suicide in a most determined and seemingly intelligent manner by taking the poison known as "Rough on Rats."

CASE IX.—Mr. —, age sixty-seven. This patient is suffering at the present time from melancholia, with marked suicidal and homicidal tendencies, slight hallucinations and great insomnia, but has no post-cervical ache.

CASE X.—Mr. —, age thirty-seven. Came to my office complaining of pain in the back of the head and neck. Something in the man's manner aroused my suspicions, and I found that he had, about six years ago, whilst living in Further India, an attack of melancholia with obstinate insomnia, slight mental confusion for a short time, and suicidal tendencies, all these symptoms having lasted about six months, and since this time he has had an aching post-cervically, which often passes away and leaves only a neuralgia over the left ear and scalp. This patient is still under treatment.

CASE XI.—Mr. —, age thirty-four, is very melancholy and apprehensive, has obstinate insomnia, sleeping about four hours with large doses of sulphonal, and has a constant drawing pain in the neck and shoulders. No suicidal tendencies. This patient is still under treatment.

CASE XII.—Miss —, age twenty-eight. Seen in consultation in a neighboring city. For about a year has had constant and great insomnia, pronounced melancholy, occasionally becoming greatly agitated and so marked that the patient would shut herself up in her room and cry

incessantly. Has constant pain in the occiput, which is at times quite severe. Has slight delusions.

CASE XIII.—Miss —, age thirty. Great melancholy for the last six months and obstinate insomnia for the same time. Has a weary aching feeling over the back and the top of the head. During the last ten years she has had several similar attacks, lasting two to three months, with insomnia, aching post-cervically and at the vertex. In one of these attacks she had a hallucination of sight, otherwise no hallucinations or delusions in any of these attacks. Has had suicidal ideas frequently suggest themselves to her, but has been able to repel them.

CASE XIV.—Mrs. —, age forty-two, wife of a physician, came to me complaining of great aching distress in the back of the head and shoulders. This led me to interrogate her closely, when I found that she had just recovered from an attack of typical melancholia with obstinate insomnia, marked suicidal and homicidal tendencies, which nothing but her strong religious feelings had enabled her to control, and which she had suffered for some six months without daring to tell her husband. This patient is still under treatment, and is almost recovered from the aching and the occasional attack of melancholy which were all that have been left of her former attack.

CASE XV.—Mrs. —, age thirty-three, sent to me by a physician. About nine months ago was attacked with melancholia shortly after severe domestic misfortune, suffered for three consecutive nights absolute sleeplessness and then began to suffer from pain in the back of the head and down the spine. Since the onset the melancholy and the ache have been present almost the whole time, but the insomnia has been treated with fair success, although she still sleeps badly and is a long time in getting to sleep. At first she had optic hallucinations occasionally.

CASE XVI.—Miss —, age twenty-six. About one year ago, after great worry and night work in nursing her mother, she began to be very sleepless, and to have an aching feeling in the back of the neck and become very melancholy. In the last three months she has heard a constant crackling sound at the back of the head. Has never had the slightest suicidal feeling.

In cases of melancholia with stupor and the melancholia attonita, I have not been able to ascertain whether or not the post-cervical ache was felt, possibly because of the stupor, but I do not think that these cases complain of it after recovery. In the cases of simple melancholia and the milder cases of agitated melancholia, the post-cervical ache is apt to last for many years in certain patients after the main disease has been entirely removed, and in some of this class it will occasionally merge into violent neuralgic attacks, during which the insomnia will return, and there may supervene some slight mental confusion and melancholia, although these recurrent attacks seldom last over a week, and are very easily treated. The insomnia is apt to be an obstinate symptom for a certain length of time in all the forms, but the cases of melancholia attonita and stupid melancholia do not suffer from insomnia after they have recovered from the main disease as much as do the cases of simple melancholia and the slighter cases of the agitated form. In the latter two classes it is not unusual to find patients suffering constantly from slight insomnia for many years, and at times becoming so sleepless as to necessitate large doses of hypnotics.

So firmly have I come to rely upon the association of this symptomatic triad that I have lately made a diagnosis in two cases by means of it. The first patient was a gentleman who came to me complaining of a distress in the back of the head and neck, which at times was painful. I learned from him that the onset dated back to six years ago, when, as he said, he had been run down and depressed. I then told him that I would outline to him his symptoms at that time, and I proceeded to tell him that he had been very very much depressed, had not been able to sleep, had thought of committing suicide, had been slightly confused in mind, and had remained in this condition for several months. He was amazed, and asked me if I was a mind reader, finally admitting that he had passed through just such an attack of melancholia, which he had concealed from everybody because he was then living in Burmah in the employ of the English Government, and was afraid that

he would lose his position if they should think him insane. The other patient was a lady who came to me complaining of the same vague distress in the back of her head and neck. I also ascertained from her that the trouble dated back some six months; and I then asked her whether she had not at the beginning been very much depressed. She answered me in the affirmative with so embarrassed an air as to make me assured that there was something concealed, and I then went on to recapitulate to her the symptoms of melancholia as I had done to the man from Burmah. She burst into tears, and admitted that she also had passed through an attack of melancholia, and astonished me in her turn by telling me that she was the wife of a well-known physician, and that she had concealed all knowledge of her mental condition from her husband, because she was afraid that he would send her to an asylum. This poor woman had absolutely on several occasions felt so strong an impulse to kill her children and herself that she had been obliged to leave the house and get away from them.

It seems to me that the knowledge of this conjunction of symptoms should be of value for both diagnostic and therapeutic reasons. Every physician in general practice, and still more every neurologist, knows how difficult it is at times to feel sure as to whether a given case of depression is one of hysteria, of grief, of depression from a gastrointestinal disorder, of depression preceding the outbreak of some other form of mental disease, or whether it is true melancholia. In the latter the suicidal and homicido-suicidal tendencies are so organic a part of the disease that the mildest case is not to be trusted, and valuable lives may be saved by some method of prompt recognition. I have no hesitation in saying that the most ghastly crimes in the annals of lunacy are those which are committed by melancholiacs who are suffering from simple melancholy, without stupor and either without delusion or hallucination or with slight tendencies of the two latter kinds, for these patients seem so reasonable and intelligent that no check is put upon them, and they attain their diabolical ends with a directness and success that is not possible to any other

class of the insane. I have known of one patient secreting for months a match under her thumb nail and a wisp of light paper in her axilla, finally to draw out the paper and light it with the end of the match and deliberately set fire to her gown so as to burn herself to death. I have known another patient, who had been watched for weeks, but who had seemed perfectly quiet and inoffensive, yet slink out of the house at midday, walk two miles to the Park, deliberately search for the most secluded part in it, remove his shirt and tear it into strips, make a rope of the pieces, and hang himself to the bough of a tree. And I could go on multiplying instances, which all of you could probably match, of women who have killed their children, of husbands who have killed their wives, of lovers who have killed the beloved, until the recital of horrors would equal the bloody reign of the Duke of Alva and the Inquisition in the Netherlands. From a therapeutic point of view the recognition of the insomnia and the post-cervical ache should enable a physician to diminish the distress and the consequent aggravation of mental symptoms.

I venture to hope that the medical profession will observe this triad of symptoms, so that we may ascertain the percentage of its presence in a greater number of cases than it has fallen to my fortune to observe.

NEW YORK, 6 E. 49TH ST.

A CASE OF CERVICAL PARAPLEGIA FROM DISLOCATION—AUTOPSY.¹

By CHRISTIAN A. HERTER, M.D.

THE following is the history of a case of cervical paraplegia from dislocation forward of the sixth cervical vertebra upon the seventh. The patient was in the Presbyterian Hospital in the service of Dr. Briddon, to whom I am indebted for an opportunity of examining the case.

J. F., a mason, fifty-seven years of age, fell head first from a scaffolding on the morning of October 9, 1889, a distance of twelve feet.

His head struck against a board, but precisely in what manner is not known.

When found the patient lay on his back in great agony. He referred his pain to his neck.

On admission to the hospital the patient was still fully conscious. Respiration was purely diaphragmatic. The pulse was good. Temperature 97.2°

A careful examination failed to elicit evidence of fracture or dislocation in any part of the cervical spine. It was noted, however, that there was great tenderness to pressure over the sixth and seventh cervical vertebræ.

The house-surgeon, Dr. Sharpe, states that when first admitted the patient was observed to have good use of his forearms and arms, although he was completely paraplegic below the upper extremities, and the fingers were weak. There were also complete anæsthesia and analgesia of the lower extremities, and of the trunk as high as the seventh cervical vertebra behind, the exact upper limit of the sensory loss anteriorly not having been noted. The loss of sensibility also involved the ulnar border of either upper extremity.

¹ Read before the New York Neurological Society, 1889.

With the exception of the plantar reflex of the left side, both superficial and deep reflexes were absent when the patient first came under observation.

Nine hours after the admission of the patient an examination was made to determine with exactness the distribution of the motor and sensory paralysis, in order to learn the upper level of the lesion in the cord.

There was complete paralysis of motion below the level of the upper extremities. In the upper extremities themselves there was complete loss of power in the intrinsic muscles of the hand and interossei, in the flexors and extensors of the wrist and in the triceps. There was slight loss of power in the pronators and supinators, and considerable weakness of the biceps. The weakness of the biceps was more pronounced on the left than on the right side; in other respects the distribution of the motor loss was highly symmetrical.

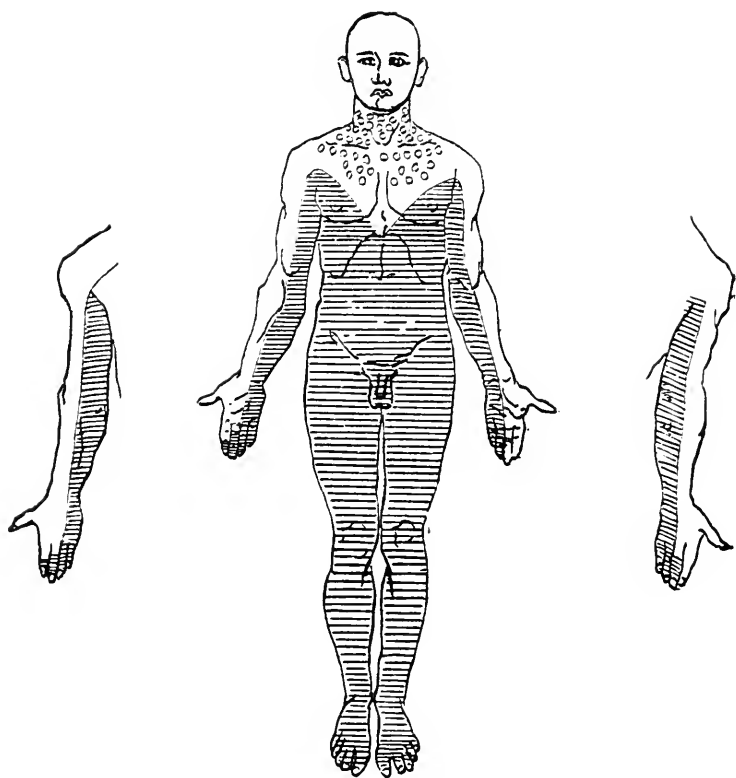
The house-surgeon stated that the weakness of the biceps at the time of this examination was distinctly greater than three hours before, and also that the muscles of the hand were involved before those of the forearm. The triceps was involved before the biceps. The pectoralis major showed some loss of power.

The deltoids, trapezii, and the muscles of the head and back were normal.


The sensory loss also presented a high degree of symmetry. The anæsthesia and analgesia involved, roughly speaking, the distribution of the ulnar, internal cutaneous and lesser internal cutaneous nerves in the arm.

On the anterior aspect of the chest the upper border of the anæsthetic area is indicated on either side by a line beginning about two inches above the axillary border and passing through the fourth rib in the mammary line to the lower end of the gladiolus.

The accompanying diagram represents with exactness the distribution of the sensory loss. It is to be noted that while on the arms and chest the symmetry is striking, on the hands there is some irregularity of distribution. On the right side the loss of sensibility involves the palmar surface



 *Area of Anæsthesia and Anelgesia.*

 *Area of Hyperalgesia.*

of the little ring and middle fingers, and of the third phalanx of the index finger. On the dorsal surface of the hand it involves only the little and ring fingers, with the second and third phalanges of the middle finger. The left hand shows loss of sensibility on the palmar aspect of the little and ring fingers and on the dorsal surfaces of the same fingers. In addition the loss involves the posterior surfaces of the second and third phalanges of the middle finger, and of the third phalanx of the index finger.

There was a cincture pain about the lower part of the neck. Just above and below the clavicles the skin was hyperalgesic.

Below the neck there was most pronounced myoedema. Fibrillary contractions could be elicited very readily below the neck, by light percussion, but the mechanical irritability of the muscles was greatest in the upper extremities.

Both deep and superficial reflexes were absent as before, and there was retention of urine.

The sense of posture was lost in the lower extremity and trunk. The temperature sense was not tested. When observed the pupils were nearly equal and of moderate size. Their reaction to light and during accommodation was not noted. The position assumed by the upper extremities is of interest, corresponding as it does very nearly with the position observed by Thorburn in cases of injury below the fifth cervical nerve-root. Both arms were slightly abducted, and the forearms tended to assume a position of flexion and supination. There was no external rotation of the arm, and when the forearm was placed in a position of full extension it remained so.

During the afternoon of the 9th the temperature rose to 104°. The pulse continued to be fair in strength. Respiration diaphragmatic.

On October 10th the patient's condition was not materially changed, except that the biceps was perhaps feebler. During the afternoon of this day the respiration became embarrassed, and at 6.30 on the morning of the 11th, the patient died from respiratory failure, having survived the accident about forty hours.

The diagnosis of the position of the upper level of the injury to the cord was based on the distribution of the motor and sensory loss. The distribution of the anæsthesia in the upper extremities corresponds closely with that attributed by Ross² and by Thorburn³ to destruction of the eighth cervical and first dorsal segments of the cord. The sensory indications, therefore, pointed to the lower end of the seventh cervical segment as the upper limit of the lesion. The indications derived from the fully developed motor palsy placed the upper level of the considerably higher in the cord, for in addition to the slight weakness developed in the pronators, supinators, and pectoralis major, there was decided though not absolute loss of power in the biceps.

According to Thorburn⁴, the biceps is represented in the fifth cervical nerve, and in Starr's⁵ recent table it is given as derived from the fourth, fifth and sixth segments of the cord. Hence, the lesion causing the fully developed paralysis was argued to have extended at least as high as the sixth segment. But, when the patient was admitted, the muscles of the hand only were parietic, and as these are supposed to be represented in the eighth cervical and first dorsal segments the indications derived from the original motor and sensory loss showed a close agreement.

The upward extension of the paralysis was so rapid and occurred so soon after the injury that it was not attributed to an ascending myelitis. Moreover, the sensory paralysis remained stationary while the motor loss ascended, and it was thought probable that the damage to the nerve elements above the eighth cervical segment was incomplete and perhaps referable to pressure from bone or to hæmorrhage into the cord, or to both, occurring soon after the original damage. In view of these considerations the upper level of the original damage was located in the seventh cervical segment.

² On the Segmental Distribution of Sensory Disorders. *Brain*, Jan., 1888.

³ Cases of Injury to the Cervical Region of the Spinal Cord. *Brain*, Jan., 1887.

⁴ Spinal Localizations as Indicated by Spinal Injuries. *Brain*, Oct., 1888.

⁵ Syringomyelia: its Pathology and Clinical Features. Table, p. 464. *Am. Jour. Med. Sci.*, May, 1888.

The autopsy, which was conducted less than twelve hours after death by Dr. Thacher, revealed a dislocation forward of the sixth cervical vertebra upon the seventh, and an oblique fracture through the left superior articular process of the sixth cervical vertebra. The extent of the forward displacement of the sixth vertebra upon the seventh was probably not more than one-third of an inch, if we may judge by the latitude of movement at the autopsy. How much pressure was being exerted on the cord at the time of the autopsy, it is impossible to say, but there seems to have been some displacement.

The vessels of the pia near the of level the dislocation were fuller than above and below, and the left anterior root of the eighth nerve was torn across. No other extra-medullary change could be detected.

The consistence of the cord was very much diminished throughout the eighth segment, and the gross appearance of the lower end of this segment was indicative of extensive disintegration of the nervous elements. The seventh segment was also decidedly soft, though less so than the eighth. Above and below these segments the consistence of the cord seemed quite normal. At the junction of the sixth and seventh segments the central canal was much distended by hæmorrhage, and this central hæmorrhage was discernible as high as the upper end of the sixth segment, but at this level there is a very slight increase only in the size of the canal.

The ascent of the motor symptoms in this case seems referable either to the hæmorrhage into the cord or to post-traumatic pressure on the seventh segment of the cord, causing sufficient damage to produce motor without sensory symptoms. If the latter mechanism be the explanation of the spread of the motor loss, it must be admitted that the sensory palsy is due entirely to the injury of the eighth segment, since the anæsthesia remained stationary. But it is perhaps more reasonable to suppose that both the seventh and eighth segments were damaged simultaneously by the original trauma. In this case the sensory loss might be referable to the injury of the seventh segment as

well as the eighth, and the subsequent motor paralysis could then be explained only on the supposition that the central hæmorrhage and slight infiltration were adequate to damage the anterior horns.

Supposing the hæmorrhage in the region where the distension of the canal is considerable, and there is some extravasation into the surrounding gray substance, to have been operative in the production of paralysis, we cannot refer any symptoms to a higher level than the junction of the sixth and fifth segments, and it is very doubtful whether the hæmorrhage observed in the upper half of the sixth segment could have been a factor.

So far, therefore, as we can draw any inferences regarding the localization of the motor and sensory functions of the cervical cord from a single case, these are, with one exception, in support of the conclusions reached by Thorburn from the study of a considerable number of cases of injury in this region. The exception relates to the representation of the biceps muscle. The fifth segment of the cord is intact in this case, and it is questionable whether the changes in the upper half of the sixth segment can be made responsible for any motor loss. Yet the biceps was all but completely paralyzed on both sides. We are thus led to the conclusion that in this case the biceps was largely represented in a part of the cord at least as low as the lower half of the sixth cervical segment.

The involvement of the intrinsic muscles of the hand before the flexors and extensors of the wrist, and of the triceps before the biceps, corresponds in a general way with the vertical arrangement of the cervical nuclei as tabulated by Thorburn. The pronators and supinators were involved before the biceps, though just when was not noted, and they were much less paralyzed than the biceps, which is supposed to have a more cephalad representation. If the nuclei were arranged in simple vertical series without horizontal differentiation or vertical overlapping, such partial immunity would probably not occur in lesions of this kind.

And upon this point we must offer a word of criticism upon Thorburn's valuable work, in that he seems to have overlooked the important horizontal differentiation which certainly exists, in an attempt to establish the vertical relationship of the nuclei. In Starr's table the extensive vertical representation of some of the muscle nuclei is taken into consideration.

I put this case upon record, not because it presents any remarkable features, but because the number of cases of this kind, with autopsy, is still small, and every isolated instance may be of some aid in the localization of future lesions of the cervical cord.

A CONTRIBUTION TO THE CLINICAL STUDY OF SPONTANEOUS DEGENERATIVE NEURITIS OF THE BRACHIAL PLEXUS.¹

By WILLIAM M. LESZYNSKY, M.D.,

Lecturer on Mental and Nervous Diseases at the New York Post-Graduate Medical School;
Attending Physician in the Department for Nervous Diseases at the Demilt Dispensary, etc.

WITHIN recent years the literature relating to peripheral neuritis has indeed been voluminous, particularly that pertaining to the multiple form. Hence, much that is definite and decided has been added to our knowledge of this affection, which, to-day, is universally recognized and firmly established.

This paper is intended to serve as a contribution to the clinical study of spontaneous neuritis affecting the brachial plexus, and is based upon the observation of the following unique case :

Michael Carney, an Irish laborer, thirty-eight years of age, first came under observation on the 15th of October, 1887, complaining of pain in the *left* shoulder, of one week's duration. He describes the character of the pain as constant, "shooting" down the inner aspect of the arm to the elbow-joint, and is apprehensive in regard to the probably serious nature of his malady, as three years ago he suffered from an attack of paralysis affecting the *right* shoulder, which began in a similar manner and resulted in six weeks of enforced idleness. His former trouble, he believes, was due to "taking cold," and the cause of his present illness he ascribes to a thorough drenching experienced in a rain-storm a few days before existing symptoms began. He has been married seventeen years, and has four children living. His wife states that she has had no miscarriages, but four of her children died from convulsions during infancy. Repeated interrogation fails to elicit any antecedent history of traumatism, syphilis, or joint disease. He confesses

¹ Read at the Annual Meeting of the American Neurological Association, June, 1889.

excessive indulgence in alcoholics, followed frequently by intoxication, but has abstained from the use of liquor during the last two weeks. Some years ago he had occasional attacks of muscular rheumatism (lumbago, pleurodynia, etc.). Bowels are constipated and appetite is poor.

Status Præsens.—Well-nourished, able-bodied man, with a moderate degree of intelligence.

Left Upper Extremity.—Motility unimpaired; muscular resistance good; no roughening or involvement of shoulder-joint; tenderness and circumscribed pain on pressure over lower portion of biceps; no objective sensory disturbance. Dynamometer registers 80 (right, 100+). All nerves and muscles react well to faradism, secondary coil, slide at 12 mm.

Right Upper Extremity.—Deltoid is markedly atrophied. Muscular resistance is excellent. He has full use of the shoulder and arm, the loss of the deltoid being replaced by the compensatory development of the other shoulder muscles. The extremity is otherwise normal. The deltoid does not react to faradism. Galvanic current of 15 milliamperes produces feeble CaCC.

Lower extremities normal. No evidence of cardiac, pulmonary, or renal lesion.

Treatment.—Abstinence from alcoholics. Blister over biceps. Calomel, gr. v; compound licorice powder, 3 i: at one dose.

Oct. 20, 1887. Reports to-day that the calomel, etc., produced free purgation, and that he has been in constant pain, which begins in the left shoulder and extends to the biceps. The pain is worse at night, and he is unable to sleep. The *left deltoid is paralyzed*, and there is some difficulty in outward rotation of humerus. No objective sensory disturbance.

Electrical Reaction.

Faradism.

L. circumflex nerve, O.

L. deltoid, 12 mm., good.

Galvanism.

O.

5 Ma.CaCC.

Ordered rest, blisters, and antipyrin twenty grains every hour for three hours, unless relief is obtained sooner.

Oct. 25th. Pain is paroxysmal and almost unendurable, darting down to the dorsum of thumb and index-finger. He is unable to sleep at night, and "walks the streets" endeavoring to obtain relief through exercise. Repeated doses of antipyrin proved ineffectual.

Slight temporary alleviation was obtained by wrapping the arm in hot-water cloths. He states that when the right arm was affected, the pain was just as severe, but did not extend below the elbow, and was always worse at night. He believes the arm and hand are getting weaker, and complains of numbness below the shoulder, extending over the radial distribution. Dynamometer registers 65 (R., 100+). There is some tenderness over the lower portion of the radial nerve, but no appreciable thickening. In the region of the cutaneous distribution of the circumflex nerve there is an area, $2\frac{1}{2}$ cm. wide and $7\frac{1}{2}$ cm. in length, where tactile, pain, and temperature senses are abolished. The numbness is greatest at this point. Temperature in mouth, $99\frac{1}{4}^{\circ}$ F.

Electrical Reaction.

Farad.

Deltoid, 12 mm., feeble.
Musculo-spiral nerve and
muscles, 15 mm., good.

Galv.

5 Ma.CaCC, slow.

He refuses to remain abed, saying that he is more comfortable while walking about. Ordered morphia sulphate, one-sixth grain, and repeat if necessary; blisters; salicylate of soda, twenty grains every three hours.

Oct. 29th. Pain was relieved by one third grain of morphia. Slept well last night without medicine. The hand is weaker. Grasp with dynamometer, 38. The biceps and triceps exhibit well-marked fibrillary tremor. In the fibres, at the lower portion of these muscles, there occur distinct, irregular, and wave-like contractions, forming "myoid tumors" parallel with the course of the muscular fibres. These circumscribed contractions are produced by either voluntary or passive motion or mechanical irritation. There is partial wrist-drop, the extensors of the forearm and fingers and the supinators being paretic. There is also

difficulty in outward rotation of the arm, indicating the involvement of the *infraspinatus* and *teres minor*.

The *pectoralis major* and the biceps group are weak, but the triceps is not affected. Area of anæsthesia unchanged. Temperature in mouth, 100° F.

Nov. 1st. Was comparatively comfortable until yesterday. Then pain began at elbow and extended over radial side of forearm to the thumb and index-finger, but is now more intense over the dorsum of thumb. Recumbency increases the pain, which is invariably worse at night. Complete *paralysis* of *infraspinatus* and *teres minor* (no outward rotation). Inward rotation of humerus good. The *pectoralis major*, *supinators* and *extensors* of forearm are also paralyzed. Triceps apparently not involved, but "jerk" is feeble. Dynamometer, 48; temperature, 99° F. Discontinue salicylate of soda. To have hydrarg. bichlorid., gr. $\frac{1}{24}$ t. i. d.

Electrical Reaction.

Farad.

Galv.

Deltoid 12 mm. feeble.

Musculo-spiral n. 15 mm.

Extensors, O.

$5\frac{1}{4}$ Ma. CaCC. feeble.

No reaction can be obtained at "Erb's Supraclavicular point" on either side.

Nov. 3d. On the night of November 1st pain was excruciating and constant, darting up and down the arm in the course of the musculo-spiral and radial nerves to the thumb and index finger. Took $\frac{2}{3}$ grain of morphia without relief. Pain began at 6 P. M., and ceased at 6 A. M. He slept well last night without morphia, and the pain has subsided. The grasp is weaker. Dynamometer 25. The *triceps* is *paralyzed* and "jerk" is abolished. There is a point of extreme tenderness on deep pressure just above the flexure of the elbow joint. Analgesia in the course of the radial distribution to the hand. Partial tactile anæsthesia, more marked over the dorsum of the thumb. Area of anæsthesia below deltoid unchanged.

*Electrical Reaction.**Faradism.*

	RIGHT.	LEFT.
Musculo-spiral N.,	15 mm. fair	O
Triceps,	"	O
Deltoid,	O	O
Extensors,	15 mm. fair	O
Median and Ulnar N. } and Muscles,	Normal.	Normal.

Galvanism.

	RIGHT.	LEFT.
Musculo-spiral N., 8 Ma. CaCC <i>slow</i> .		O
Triceps, 6½ Ma. CaCC > AnCC.	3½ Ma. CaCC > AnCC <i>D.T.</i>	
Deltoid, 15 " CaCC feeble.	3 " CaCC > AnCC <i>slow</i> .	
Extensors 7 " CaCC > AnCC.	3½ " " " "	
Median and Ulnar N. } and Muscles,	Normal.	Normal.

Nov. 5th. No pain since last note. Only slight aching in elbow. Grasp is stronger. Dynamometer 38. General condition of extremity unchanged. During the last few days he felt occasional slight pain, accompanied by tremor over the right serratus magnus.

*Measurement.**Circumference.*

	RIGHT.	LEFT.
Arm, 6 in. below acromion (extremity pendent)	10¾ in.	10½ in.
Forearm, 4 in. below olecranon (" semi-flexed)	10½ "	10½ "

Nov. 8th. Slept well on the 5th without morphia. Pain returned the following night. *Causalgia* affecting the dorsum of hand during the past week. On the 7th, had pain all day, mostly at the flexure of the elbow-joint, over the lower end of the biceps, with continued numbness in the course of the radial nerve. Last night suffered from severe pain, stabbing, shooting and darting in character. He obtained some relief from the application of hot-water cloths. There is a slight œdema over the dorsal aspect of hand.

Internal rotation of humerus abolished (indicating involvement of the subscapular nerves). Dynamometer 32. Sensory disturbance, etc., same. No pupillary symptoms.

Nov. 10. Pain has extended over the forearm. Anal-

gesia and partial tactile anæsthesia over the radial and external cutaneous branches of the musculo-spiral nerve. *The biceps is paretic.* With great effort he succeeds in slightly flexing the forearm. Pain is aggravated by passive extension of the forearm, owing to deep-seated inflammation, probably of the musculo-spiral nerve. Slight pressure at the flexure of the elbow produces severe pain, which radiates over the extensors. (I was unable to satisfactorily determine the existence of thickened or swollen nerve).

Electrical Reaction.

	<i>Farad.</i>	<i>Galv.</i>
External Cutaneous N.,	12 mm.	6 Ma. CaCC.
Biceps Group,	" fair.	" CaCC=AnCC.

Nov. 19th. Did not appear since last note until to-day. Remained abed until yesterday as he felt weak. No pain. Only numbness, as usual. Slept well. Upon leaving his bed, the pain returned in the shoulder (stormy weather). Pain is paroxysmal, and was so severe last night that he was unable to sleep. Constant causalgia over the hand and the wrist-joint. When the paroxysm of pain comes on, it is accompanied by "weakness" in the præcordial region, with a feeling of faintness, and then "doesn't care whether he dies or not." Is relieved by a full inspiration and "fixing" chest. No pupillary symptoms. Scapular muscles and deltoid undergoing atrophy. Biceps group weaker. Flexion of hand and fingers good. Oedema over the dorsum of the hand increasing. Dynamometer 33. Analgesia over the entire course of the external cutaneous and radial nerves, with partial tactile anæsthesia and loss of temperature sense.

Electrical Reaction.

	<i>Farad.</i>	<i>Galv.</i>
Musculo-spiral nerve,	O	O
Triceps,	O	5 Ma. CaCC > An CC <i>slow.</i>
Deltoid,	O	5 " AnCC > CaCC <i>feeble.</i>
Extensors,	O	5 " CaCC > AnCC <i>slow.</i>
Biceps Group,	12 mm. feeble.	
Supra and infraspinati,	O	

Median and ulnar nerves and muscles react well to faradism 17 mm.

Continue hydrarg. bichlorid $\frac{1}{4}$ t. i. d.

Nov. 22d. Location and severity of pain unchanged. *Biceps group paralyzed.* Dynamometer 30.

Measurement. Circumference of arm $10\frac{1}{4}$ in. Forearm $9\frac{1}{2}$ in. Loss, $\frac{1}{4}$ and 1 in.

Electrical Resistance. } Right upper extremity, 1700 ohms.
See p. 33 } Left " " 2280 "

Being 580 ohms greater in the affected arm.

Nov. 26. He was obliged to take two doses every night to relieve the pain, which also continues during the day. This morning he awoke with pain, as usual, which was worse in the course of the radial nerve, and affected the thumb and index finger. Causalgia continues. Trophic changes in the course of the radial distribution over the thumb and index finger. The skin is pale, glossy, œdematous and anæsthetic. Dynamometer 20. Atrophy of scapular muscles and extensors of forearm increasing. Anæsthetic area unchanged. Extremely sensitive point over the pectoralis major muscle on a line three inches above the nipple. *The muscles supplied by the median and ulnar nerves are not affected.*

Electrical Reaction.

	<i>Farad.</i>	<i>Galv.</i>
Extern. Cutan. N.	$13\frac{1}{2}$ mm. <i>slow.</i>	$6\frac{1}{2}$ Ma. CaCC=An CC.
Biceps Group,	" "	" "
Median and Ulnar Nerves (and Muscles),	Normal.	

Dec. 1st. During the last three nights has been free from pain, which is now confined to the dorsum of the thumb and index-finger. Dynamometer, 22. Extreme tenderness over biceps and pectoralis major.

Electrical Reaction.

	<i>Farad.</i>	<i>Galv.</i>
Mus. spiral nerve, - -	O.	O.
Triceps (scap., head), -	O.	6 Ma. CaCC, slow.
" (short, head), -	O.	6 Ma. AnCC > CaCC, slow.

Extensors, - - - - O.	8½ Ma.AnCC>CaCC, slow.
Scapular muscles, - - O.	6 Ma.CaCC>AnCC, “
Deltoid, - - - - O.	6 Ma.AnCC>CaCC, “
Biceps group, 13½ mm. (fair).	9 Ma.AnCC>CaCC, “
Median and ulnar nerves and muscles, 14 mm., - - -	5½ Ma.CaCC, <i>normal</i> .

Dec. 10th. Very little pain until this 3 A. M. (raining). Causalgia constant. Some ability in inward rotation. No outward rotation. Muscular atrophy increasing. Sensory disturbance same. Some hyperæsthesia in palm. No change in motility. Dynamometer, 20. Tenderness over the biceps and pectoralis major unchanged. Administration of hydrag. bichlorid., gr. $\frac{1}{24}$, t. i. d., continued.

Electrical Reaction.

<i>Farad.</i>	<i>Galv.</i>
Musc. spiral nerve, - O.	O.
Triceps, - - - - O.	10 Ma.CaCC>AnCC, slow.
Extensors, - - - - O.	6½ “ “ “
Deltoid, - - - - O.	10 “ “ “

Dec. 17th. Tenderness over the pectoralis major and biceps has disappeared. Otherwise unchanged. Dynamometer, 23.

Electrical Resistance.—Right = 5590 ohms. Left = 6480 (dif. = 890).

Electrical Reaction.

<i>Farad.</i>	<i>Galv.</i>
Musc. spiral nerve, - - O.	O.
Triceps, - - - - O.	6 Ma.CaCC>AnCC, slow.
Extensors, - - - - O.	“ CaCC=AnCC, “
Deltoid, - - - - O.	“ CaCC>AnCC, “
Biceps group, 15½ mm. (slow), - - - - -	“ AnCC>CaCC, “
Median and ulnar nerves, 13½ mm., - - - - -	“ CaCC, <i>normal</i> .

Measurement.—Circumference, arm, $9\frac{3}{4}$ in.; forearm, $9\frac{1}{2}$ in. (loss, $\frac{1}{2}$ in.).

Dec. 29th. Some improvement in the strength of the biceps. Fibrillary contractions and tenderness over biceps. No other change.

Electrical Resistance.—Right = 5400 ohms. Left = 6300 (dif. + 900).

Electrical Reaction.

	Farad.		Galv.
Musc. spiral nerve, - - O.	O.		
Triceps, - - - - - O.	8 Ma.CaCC > AnCC, slow.		
Extensors, - - - - - O.	4 " = " "		
Deltoid, - - - - - O.	8 " " " "		
Biceps group, 14 mm. (slow).	5 " " " "		
Scapular muscles, - - O.	8 " > " "		

At his request he was sent to Bellevue Hospital.

Feb. 7, 1888. He left the hospital to-day. Since last note had very little pain, which was limited to the hand. He has regained some power in the arm. The only perceptible improvement is in the biceps group. Area of analgesia has diminished in the district of the circumflex, but is still well defined. Tactile sensibility improved. No loss of muscular sense. Trophic changes in the hand are markedly diminished. Atrophy of the scapular muscles increasing. General health good. Sleeps well. Shoulder-joint relaxed and articular surfaces roughened.

Electrical Reaction.

	Farad.		Galv.
Musc spiral nerve, - - - O.	O.		
Triceps, - - - - - O.	8 Ma.AnCC > CaCC, slow.		
Extensors, - - - - - O.	5 " CaCC > AnCC, "		
Deltoid, - - - - - O.	8 " AnCC = CaCC, "		
Biceps group, - - - 12½ mm.	4½ Ma.CaCC > AnCC, "		

Feb. 9, 1888:

Electrical Resistance.—Right = 5200 ohms. Left = 7000 (dif. + 1800).

March 10th. Since last note suffered occasional nocturnal paroxysmal pains below elbow-joint in radial distribution. He has been a flagman on the Long Island Railroad during the last three weeks. Exercising the affected

arm produces swelling and pain over the wrist-joint and soreness over the pectoralis major. General health good. Has abstained (?) from the use of liquor. Atrophy of shoulder muscles has increased. No improvement in deltoid, triceps, or extensors of forearm. Biceps group improving. Dynamometer (stiffer spring), L. = 27; R. = 100 +. Area of anæsthesia decidedly diminished above elbow-joint. No loss of muscular sense. No trophic disturbance save in dorsal aspect of thumb. Roughening of articular surfaces of shoulder-joint.

Electrical Resistance.—R. = 4700 ohms. L. = 6300 (dif. + 1600).

Electrical Reaction.

<i>Farad.</i>	<i>Galv.</i>
Musc. spiral nerve, - - - O.	20 Ma. produces feeble CaCC in triceps.
Triceps (outer head), 15½ mm. (feeble), - - - - -	
Extensors, - - - - - O.	8 Ma. CaCC > AnCC.

Measurement.—Circumference, arm, 9¼ in.; forearm, 9 in.

April 14th. Complains of sudden tremors, localized in spots over the arm and extending to the hand. He says the same thing occurred in the other hand before recovery. No outward rotation of humerus. Very feeble inward rotation. The only improvement in motility is in the biceps group. With great effort he succeeds in producing partial flexion of forearm. No vaso-motor disturbance. No trophic changes in hand. Tactile anæsthesia in spots in the course of the radial nerve only. Area of analgesia limited to two or three spots over the radial distribution at the wrist. Shoulder-joint not so rough as at last examination.² Atrophy of scapular muscles unchanged. Dynamometer, 30.

² He was recently shown by me at a meeting of the New York Neurological Society (April 3, 1888), at a time when the shoulder-joint was "roughened," owing to paralysis of the shoulder muscles and consequent disuse of the limb. This led one of the members present to suppose that this condition of the joint may have existed prior to the development of the neuritis.

Electrical Resistance.—Right = 5500 ohms. Left = 4330 (dif. — 1170).

Electrical Reaction.

<i>Farad.</i>	<i>Galv.</i>
Musc. spiral nerve, - - - O.	O.
Supra- and infraspinati, - - O.	7 Ma. CaCC, feeble.
Deltoid, - - - - - O.	7¼ Ma. CaCC=AnCC, slow.
Triceps, - - - - - O.	6 " " " "
Biceps group, - - - 14½ mm.	5½ " " " "
Extensors, - - - - - O.	9 " " " "
Median and ulnar nerves and muscles, - - - - 14½ mm.	Normal.

April 28th. *Electrical Resistance.*—Right = 5400 ohms. Left = 4500 (dif. — 900).

June 7th. No pain during the last three months. Dynamometer, 33. Roughened shoulder-joint and atrophy unchanged. Slight improvement in inward rotation. No outward rotation. Biceps group have recovered with good resistance to passive motion. Supination of hand is accomplished by the biceps only. No other improvement in motility. The dorsum of the hand is œdematous. Anæsthesia is limited to one small spot over the dorsum of the thumb. The entire surface of the extremity is perspiring, save over the radial distribution below the wrist, where the skin is dry.

Measurement.—Circumference, arm, 9¼ in.; forearm, 9¼ in.

Electrical Reaction.

<i>Faradism.</i>
Biceps, - - - - - 15 mm.

<i>Galvanism.</i>
Deltoid, - - - - - 7 Ma. CaCC>AnCC, slow.
Triceps (scap. head), - - - 12 " AnCC=CaCC, "
Triceps (short head) - - - 8 " CaCC>AnCC, "
Biceps group, - - - - - 5 " " " " "
Extensors, - - - - - 8 " AnCC>CaCC, "

Sept. 11th. In rainy weather has occasional pain and numbness in the hand and over the deltoid. Atrophy of scapular muscles, deltoid, triceps, and extensors of forearm. Complete paralysis of the deltoid and extensors. Partial paralysis of the triceps. Inward rotation good. No outward rotation. Biceps group normal. Dynamometer: L., 43; R., 100+. No vaso-motor changes. No sensory disturbance. Area of anæsthesia has disappeared. Roughening at shoulder-joint unchanged. Elbow-joint normal. Complete relaxation of wrist-joint. No roughening.

Measurement. Circumference, arm $9\frac{1}{2}$ in.; forearm, $8\frac{3}{4}$ in.

Electrical Reaction.

	<i>Farad.</i>	<i>Galv.</i>
Musc. cutan. nerve } and biceps group }	10 mm.	4 Ma. CaCC.
Supraspinatus, - O.		5 Ma. CaCC. (slow).
Infraspinatus, - O.	10	" O.
Triceps (short head, 10 mm.	7	" AnCC>CaCC. (slow).
" (scap.head), O.	10	" " " "
Deltoid, - - - O.	7	" AnCC=CaCC. "
Extensors, - - - O.	7	" AnCC>CaCC. "

Median and ulnar nerves and muscles normal.

Dec. 27. Since last note he has been at work driving a horse. The atrophied muscles are improving. Inward rotation good. Partial outward rotation and improvement in the action of the pectoralis major. There is a slight return of power in the deltoid. Has good use of the triceps. Biceps group have remained well. There is a tender point over the radial nerve, producing radiation of pain to the tip of the thumb and index finger.

Measurement. Circumference, arm $9\frac{3}{4}$ in.; forearm, $9\frac{1}{8}$ in.

Electrical Reaction.

	<i>Farad.</i>	<i>Galv.</i>
Musc. spiral N, - - O.		O. - - - - -
Supraspinatus, - $10\frac{1}{2}$ mm.		- - - - -
Infraspinatus, - O.		16 Ma. CaCC, (feeble).
Deltoid, - - - $10\frac{1}{2}$ mm.		10 " AnCC>CaCC (slow).
Triceps, - - - "		10 " CaCC=AnCC.
Biceps group, - "		- - - - -
Extensors, - - O.		20 Ma. No polar reaction ; only feeble reaction to labile cathode.

Median and ulnar nerves and muscles normal.

Jan. 31, 1889. No pain since last note. After using the left hand and arm, numbness and fibrillary contractions occur over the dorsum of the hand. The motility is increasing in the shoulder and upper arm muscles. No improvement in the extensors and supinators of forearm.

June 15. He has been steadily employed since last note, and the condition of the left upper extremity is much better. The atrophied muscles are improving. Inward rotation of the humerus is good. Outward rotation is incomplete. The deltoid muscle is weak, but its motility is restored. Triceps and biceps group normal. There is some return of voluntary power in the extensors and supinators of forearm and in the extensor communis digitorum. There is slight roughening of the shoulder-joint.

Measurement. Circumference, arm $10\frac{1}{4}$ in.; forearm, $9\frac{1}{2}$ in.

Electrical Resistance. R. = 8700 ohms; L. = 6300 (dif. —2400).

Electrical Reaction.

<i>Farad.</i>	<i>Galv.</i>
Infraspinatus, 1 mm. (feeble).	$22\frac{1}{2}$ Ma. CaCC>AnCC (feeble)
Deltoid, - 13 mm.	$8\frac{1}{2}$ " " "
Mus. spiral N, 1 mm. (feeble).	$12\frac{1}{2}$ " CaCC (feeble).
Triceps, - 13 mm.	- - - - -
Biceps group, "	- - - - -
Extensors, - O.	19 Ma. AnCC>CaCC (feeble).
Median and ulnar nerves normal.	

During the entire period of observation he was extremely irregular in his attendance. Many times it was necessary to send for him to appear for examination. He virtually received no systematic treatment. The only instructions known to have been indifferently followed at home, after acute symptoms had subsided, were passive motion to all joints, daily bathing and manipulation of the muscles, and keeping the limb suspended and protected from exposure.

SUMMARY.

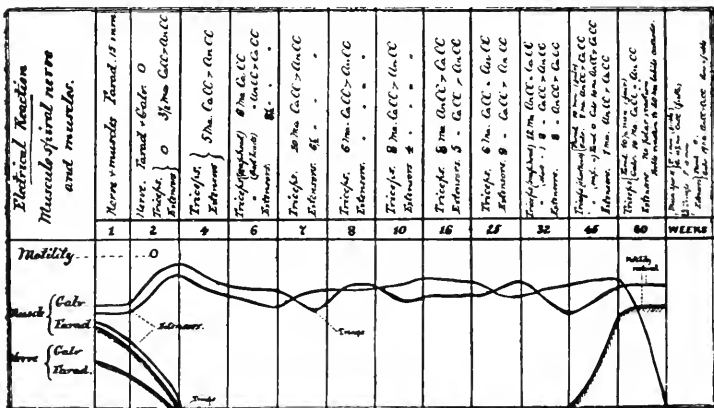
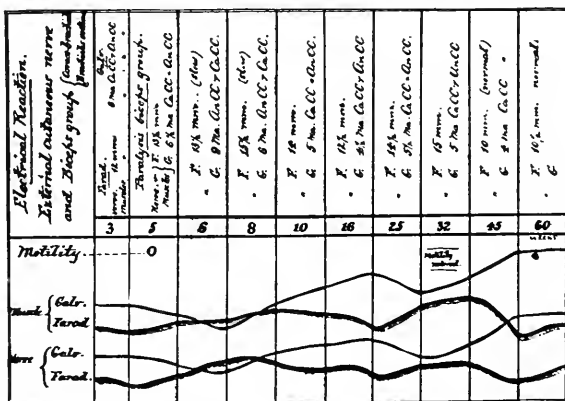
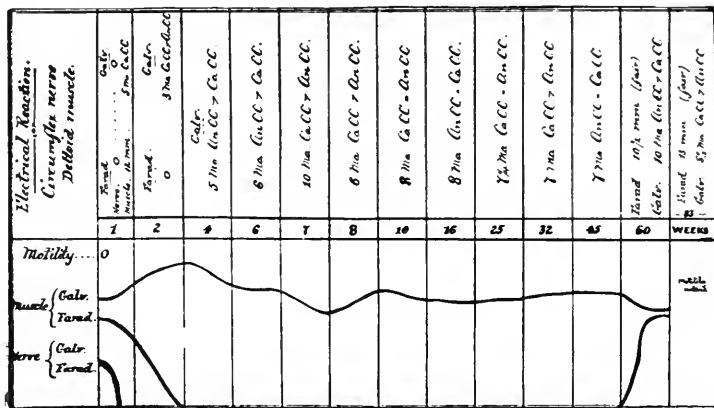
A man, thirty-eight years of age, shortly after exposure to cold (without antecedent history of joint disease or injury), suffered from severe paroxysmal pain in the left shoulder, rapidly followed by paralysis of the deltoid. After short but varying intervals of freedom from acute pain, another paroxysm would occur, accompanied by additional paralyses. These attacks, extending over a period of four weeks, involved all of the muscles innervated by the circumflex, suprascapular, subscapular, musculo-cutaneous and musculo-spiral nerves. There was anæsthesia in the domain of the circumflex, external cutaneous and radial nerves. Well-marked atrophy, with the reaction of degeneration, existed in all of the paralyzed muscles.

Trophic changes were present in the skin over the thumb and index finger.

PARALYSES.	Electrical Resistance.			MEASUREMENTS.		
	RIGHT. LEFT.			ARM FOREARM		
Oct. 20 th '91 [1st day] { DELTOID TERES MINOR.				CIRCUMFERENCE	RIGHT 10 $\frac{1}{2}$ inches 10 $\frac{1}{2}$ inches	
Nov. 1 st . [24 th] { SUPRASPINATUS INFRASPINATUS PECTORALIS MAJOR SUPINATORS EXTENSORS	Nov. 22 nd '91	1700	2280	Octms (+ 570)	Nov. 5 th '91	
	Dec. 11 th	5590	6430	" (+ 890)	Nov. 22 nd	" 10 $\frac{1}{2}$ " 9 $\frac{1}{2}$ "
	Dec. 29 th	3400	6300	" (+ 500)	Dec. 11 th	" 9 $\frac{1}{2}$ " 8 $\frac{1}{2}$ "
Nov. 3 rd . [26 th] { TRICEPS	Feb. 9 th '92	5200	7000	" (+ 1800)	March 10 th '92	" 9 $\frac{1}{2}$ " 9 "
Nov. 8 th . [31 st] { SUBSCAPULARIS TERES MAJOR LATISSIMUS DORSI	March 10 th	8100	8300	" (+ 1800)	June 17 th	" 9 $\frac{1}{2}$ " 9 $\frac{1}{2}$ "
	April 14 th	5590	4330	" (- 1170)	Sept. 11 th	" 9 $\frac{1}{2}$ " 8 $\frac{1}{2}$ "
Nov. 22 nd . [45 th] { BICEPS CORACO-BRACHIALIS BRACHIALIS-ANTICUS	April 28 th	5400	9500	" (- 900)	Dec. 22 nd	" 9 $\frac{1}{2}$ " 9 $\frac{1}{2}$ "
	June 15 th '92	8100	6300	" (- 2800)	June 15 th '92	" 10 $\frac{1}{2}$ " 9 $\frac{1}{2}$ "

TABLE A.

The median and ulnar nerves were not implicated. Almost complete recovery at the end of two years. It is well known that motor function is always more easily abolished than sensory function, and that in cases of recovery from damage sensation invariably returns before motion. In this instance, the area of anæsthesia began to diminish after four months. At the end of one year all disturbance of sensibility had disappeared.



The return of motility took place in the following order, the muscles attacked last being the first to recover (see table).

- 1st. Biceps group.
- 2d. Subscapularis and teres major.
- 3d. Triceps.
- 4th. Supraspinatus, infraspinatus, teres minor, deltoid.
- 5th. Extensors and supinators of forearm.

THE ELECTRICAL RESISTANCE.

In obtaining the electrical resistance in the extremity, the following method was adopted: A flat flannel-covered sheet-lead electrode, 2 x 4 inches, being thoroughly moistened with hot salt water, was securely attached over the nucha. A similar electrode, 1 x 2 inches, was firmly fastened in the palmar surface of the hand, the hands and the electrode having been previously soaked in the hot salt solution.

A sufficient number of Leclanché elements were then slowly introduced in the circuit (descending current) until 4 milliampères were registered on the meter. The same process was then applied to the other arm.

Subsequently, the amount of resistance was determined by means of a wire rheostat. A sufficient number of ohms (in place of the patient's body) were introduced in the circuit produced by the same electromotive force used in the examination of the patient until the meter registered 4 milliampères.

The stationary electrode at the neck was kept thoroughly wet. In order to produce uniform pressure in both hands, and to avoid unequal condensation of tissue, the electrode was retained in position by a band.

As there are so many elements of error to be eliminated before we can succeed in obtaining an accurate measurement of the electrical resistance in the human body, I would only claim the measurements in this case to be approximately correct.

In all eight measurements were made, extending over a period of nineteen months (see table A.) Five comparative examinations, during the existence of acute symptoms, invariably revealed a condition of greater resistance in the affected limb. At three subsequent examinations, after the subsidence of all acute symptoms, the resistance was found markedly diminished.

These uniform results are certainly conclusive in establishing the fact that the electrical resistance was increased during the active stage of the disease, and diminished during convalescence.

It will be noted that on November 22d, 1887, the resistance was $R=1700$ ohms, left 2280; while on June 15th, 1889, it was $R=8700$ ohms; Left $=6300$.

That such an apparent discrepancy should exist, can only be explained (in the last observation) on the ground of the probably insufficient moisture of the skin, or the difference in the pressure of the electrodes as compared with previous examinations. Despite this great difference in the resistance from time to time, the uniformity of the comparative variations, at each measurement, is sufficiently suggestive to warrant further investigation along this line. These observations are merely tentative, and under existing circumstances cannot be considered of diagnostic significance.

Before analyzing the salient features of this case, it would be well in this connection, to present a brief resumé of our knowledge of the anatomy and physiology of the brachial plexus. It is beyond the scope of this paper to enter at length upon a discussion of this subject.

Quite a diversity of opinion seems to exist as to the gross anatomy of the brachial plexus. Gray³ states, that "the fifth and sixth cervicals unite near their exit from the spine into a common trunk; the seventh cervical joins this trunk near the outer border of the middle scalenus; the three nerves thus form one large single cord. The eighth cervical and first dorsal nerves unite behind the anterior scalenus into a common trunk. Thus two large trunks are formed,

³ Anatomy Descriptive and Surgical, 1871, p. 638.

the upper one by the union of the fifth, sixth and seventh cervicals; and the lower one by the eighth cervical and first dorsal. Opposite the clavicle, and sometimes in the axilla, each of the cords gives off a fasciculus, by the union of which a third trunk is formed."

Heath, Leidy, Quain, Ellis and Flower give the same arrangement, while the arrangement furnished by Sappey, Cruveilhier, Hirschfeld, Henle, Hyrtl, Longet and Lucas differs mainly from the preceding in that the seventh does not join the common cord of the fifth and sixth, but runs as a separate trunk, dividing below as do the other trunks. (Walsh). Other writers to the number of thirty hold different views in regard to the formation of the plexus.

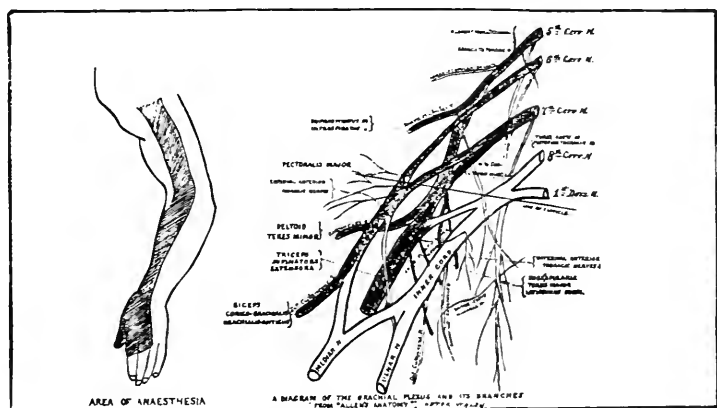


CHART B.

According to the researches of Walsh⁴ the arrangement of the plexus is not so variable, as most of the variations can be artificially produced. As a result of his examination of 350 plexuses, he concludes that "nearly every plexus will be found to resolve itself into one and the same arrangement, (see chart B) and it will also be discovered that most of the numberless variations to be found in anatomical works are nothing but normal arrangements distorted by wrong dissections." In regard to the distribution of the filaments composing the five primary trunks to

⁴ Amer. Jour. Med. Sciences, Oct. 1887.

the terminal branches of the plexus, the same writer makes the following statement, based upon the study of 74 plexures dissected after maceration in dilute nitric acid.⁵

The *musculo-cutaneous* was supplied by the fifth and sixth in 50 cases, in 23 by these and the seventh, and in one (an anomalous plexus) by a few filaments from the fourth and by the fifth and sixth. The *median* in 66 by all five, and in 8 by the four lower. The *ulnar* in 71 by the seventh and eighth and first dorsal, in two by the two latter, and in one (same anomaly as above) almost entirely by the seventh a few fibres being furnished by the eighth. The *Circumflex* in 63 by the fifth and sixth, in ten by the fifth, sixth and seventh, and in one (same anomaly) by the seventh and eighth.

The *musculo-spiral* in 67 by the fifth, sixth, seventh and eighth. In 6 by all five, and in one (same anomaly) by the sixth and seventh.

The result of Herringham's⁶ dissection of the brachial plexus in the human fœtus and in the adult differs only slightly from that of Walsh. He claims that "the *median* nerve is formed by two heads, into the outer the sixth and seventh always enter, while the fifth does not. The inner is formed always by branches of the eighth and ninth,⁷ sometimes with the addition of some bundles of the seventh. The median then is made of the sixth, seventh, eighth and ninth. The sixth bundle runs down the outer side of the nerve from the top to the bottom. The supply of the fifth by its anterior branch ends therefore with the musculo-cutaneous nerve. The eighth and ninth usually supply the flexor sublimis and always the deep flexors. After the forearm muscles have been supplied, the remainder of the median which comes from under the flexor sublimis always contains fibres from the sixth, seventh and eighth roots and sometimes a bundle from the ninth. The most common origin

⁵ Minute dissection and maceration of the brachial plexus in dissociating liquids was done by W. Krause many years ago. *Beitrag zur Neurologie der oberen Extremität*, 1865.

⁶ *Proc. Roy. Soc. Lond.* 1886--xli- 423.

⁷ He calls the first dorsal nerve the ninth spinal root.

of the *ulnar* nerve is from the eighth and ninth together. The *circumflex* is derived from the fifth and sixth alone. The *musculo-spiral* is sometimes formed by all four upper roots, usually by the sixth, seventh and eighth alone.

From a physiological standpoint, the experiments of Ferrier and Yeo⁸ command attention. They found that in the brachial plexus in monkeys, no *ulnar* movement occurred when any of the anterior roots above the first dorsal were stimulated. No *musculo-cutaneous* movement followed stimulation of any roots below the sixth cervical; but both *median* and *musculo-spiral* movement followed stimulation of the sixth, seventh and eighth cervical and first dorsal nerves.⁹

Gowers¹⁰ expresses the opinion that we are not justified in transferring these facts to man, except in so far as they receive confirmation from human anatomy and pathology. Nevertheless, much has been added to our knowledge of the function of the brachial plexus by the study of tumors, such as neuromata, and surgical as well as other injuries affecting its branches, which to a great extent favors the confirmation of Ferrier's experiments.

The clinical observations of Erb¹¹ have served to throw additional light upon the localisation of lesions in the brachial plexus. In the year 1874, he described a form of paralysis arising spontaneously as well as from traumatism, affecting simultaneously the deltoid, biceps, brachialis anticus and the supinator longus. Occasionally the supinator brevis, and at times all the muscles supplied by the median nerve are coincidently involved. The first-named group of muscles, however, are always chiefly if not exclusively affected. He believes the seat of the lesion to be at a point where the fibres forming the circumflex, musculo-cutaneous and a part of the musculo-spiral nerves lie in close proximity to each other. He further states that it is

⁸ Proc. Roy. Soc. Lond. 1881, xxxii.

⁹ Ferrier has since stated (Proc. Roy. Soc. Lond. 1883—xxxv) that the relations he gave were all one nerve too high. I have therefore designated the nerves in accord with his note of correction.

¹⁰ Dis. Nerv. Syst., Am. Ed. 1888, p. 86.

¹¹ Ziemssen's Cyclop. 11, p. 561.

possible, by very careful Faradic excitation of the several branches of the plexus, to succeed in discovering in many individuals, a spot (which corresponds to about the point of emergence of the sixth cervical nerve between the *scaleni*) from which the deltoid, the biceps, *brachialis anticus* and the *supinator longus* may be thrown into common and very energetic contraction.¹²

He is consequently of the opinion that this form of paralysis has its seat in the root of the brachial plexus at this point. Hoedemaker¹³ however, maintains that the lesion is situated in the cord formed by the fifth and sixth cervical nerves, and calls attention to the fact that these two nerves, owing to their superficial position in their exit between the *scaleni* muscles, are especially exposed to injury.

Additional cases of Erb's paralysis have been recorded by Remak,¹⁴ Bernhardt,¹⁵ Weill,¹⁶ Nonne,¹⁷ Lannois,¹⁸ Giran-deau,¹⁹ and others.

From our knowledge, based upon the foregoing anatomical, physiological and clinical data, we are enabled to determine, with some degree of accuracy, the seat of the pathological process in our case.

The distribution of the paralysis corresponds with a lesion limited to the anterior branches of the fifth, sixth and seventh cervical nerves. The escape of the ulnar nerve and the absence of all pupillary phenomena, are indicative of the preservation of the two lower roots of the plexus.

The elaborate experimental and clinical studies of Klumpke²⁰ show that oculo-pupillary troubles occur in total paralysis of the brachial plexus, but only in those cases

¹² I have repeatedly demonstrated this "supraclavicular point," which is one of the most interesting in electro-diagnosis.

¹³ *Deutsch Archiv. Klin. Med.* v 40, p. 62, 1887.

¹⁴ *Berlin Klin. Woch.* No. 9—1877.

¹⁵ *Zeitsch. fur. Klin. Med.*, Bd. iv. 3 Heft.—1882.

¹⁶ *La Province Medicale*, No. 48—1888.

¹⁷ *Deutsch Arch. Klin. Med.*, V. 40, p. 62—1887.

¹⁸ *Revue de Medicine*, p. 988—1881.

¹⁹ *Ibid.* p. 186—1884.

²⁰ *Revue de Medicine*, 1885—v— p. 739.

where the lower roots are involved, and arise from a lesion affecting a communicating branch of the first dorsal nerve.

The independent investigation of both Herringham and Walsh show that the *median nerve* invariably received fibres from the sixth and seventh cervical nerves. Its escape, therefore, is certainly extraordinary, and can only be accounted for on the ground of its anomalous origin.

It would be difficult to reconcile these facts upon any other hypothesis.

Cases of this character must either be of very infrequent occurrence, or they are not reported, as no analogous instance can be found in medical literature.

The nearest approach to analogy is the following history, reported by J. Straus.²¹ A vigorous, muscular man, 33 years of age, upon awaking one morning, without any antecedent history of injury, rheumatism or exposure, found the right hand numb and the arm seemed heavier than usual. Within two days the numbness had extended to the entire arm, with gradual increase of weakness, until all of the muscles of the extremity were paralyzed, save those innervated by the median nerve.

A study of the sensibility confirmed these facts. Anæsthesia existed in the cutaneous distribution of the circumflex, internal cutaneous, musculo-spiral, ulnar and musculo-cutaneous nerves, while sensation was preserved in the entire domain of the median. The paralyzed muscles reacted to faradic and galvanic irritation.

The patient made a complete recovery at the end of seven weeks. The nerves regained their function in the following order:

1st. Musculo-cutaneous (within two days.) 2. Musculo-spiral. 3d. Internal cutaneous. 4th. Ulnar. 5th. Circumflex.

The return of sensation and motility in the course of the musculo-spiral and the ulnar was parallel and simultaneous. It was only in the circumflex that the return of motion pre-

²¹ "Note sur un cas de paralysie spontanéé du plexus brachial (avec intégrité du nerf median) et sur quelques localisations rarer de paralysie du plexus brachial." *Gaz. Hebdom.*, 1880, No. 16.

ceded that of sensibility. He believed the case to be one of congestive or inflammatory origin, affecting the branches of the brachial plexus. He offered no explanation for the intact condition of the median nerve, and claimed that no analogous case had been reported.

Buzzard²² refers to a case (without history) of neuritis affecting certain branches of the brachial plexus and occasioning local paralysis, exquisite pains, hyperalgesia, muscular atrophy, abolished or diminished electrical excitability, and trophic changes in the skin.

Althaus²³ instances the case of a girl sixteen, in whom, after ten days of severe pain and numbness in the right hand and arm, the entire extremity became completely paralyzed. This was accompanied by anæsthesia, trophic changes in the skin, and loss of faradic irritability. There was no atrophy.

Diagnosis.—Rheumatic neuritis of the brachial plexus. Recovery from periphery to centre at the end of seven months.

THE ETIOLOGY.

The prominent factors leading to the development of the neuritis in my case, were alcoholism as the predisposing element and exposure to cold the exciting cause. This may or may not have been accompanied by over-exertion. Leyden²⁴ maintains that the spontaneous or primary form of multiple neuritis is most commonly caused by exposure or over-exertion, and frequently by both combined. The same may be said of the form of neuritis now under consideration. It is evident that these two factors play an important part in the development of the so-called idiopathic neuritis, as they do in diseases of the spinal cord. According to Caspari's observations, cold is one of the most frequent causes of neuritis in Russia, where remarkable temperature variations exist during many months. Since Magnus Huss in 1852 directed attention to a form of paralysis occurring in alcoholic subjects, many important

²² Lond. Lancet, 1885, vii., p. 983.

²³ Med. Chir. Trans., Lond., 1871, v., 54.

²⁴ Die Entzündung der peripheren Nerven, Berlin, 1888.

contributions to our knowledge of this matter have been made, so that it now is a well-known and established fact that the toxic effect of alcohol upon the peripheral nerves renders them more vulnerable to affections of an inflammatory or degenerative nature, and that frequently the nerve-trunks become similarly implicated.

PROGNOSIS.

The power of regeneration of nerve-fibres seems almost unlimited, the length of time required for the completion of the regenerative process varying from a few weeks to seven years or more. Poore²⁵ refers to a patient with traumatic paralysis of the brachial plexus, accompanied by vaso-motor and trophic changes, who had almost completely recovered after four years and a half.

In one of Trepte's²⁶ cases of neuritis migrans, recovery took place at the end of seven years.

The probable duration and progress of the case can only be determined with any degree of accuracy by careful and repeated electrical examination.

The electrical reactions in the nerves and muscles, as pointed out by Erb,²⁷ are of the greatest prognostic significance. He says: "Under otherwise similar circumstances, *i. e.*, in one and the same form and cause of disease, the lesion is so much more serious, the duration of the disease the longer, the chance of complete restitution slighter, the more developed and complete the De R. is, and the more advanced the stage in which it is found. Partial De R. is therefore more favorable than the complete, the later stages more unfavorable than the earlier.

In the cases of brachial plexus paralysis reported by Remak,²⁸ the brachialis anticus and biceps recovered first and the supinator longus last. He is therefore of the opinion that this may perhaps be dependent upon the length of the nerves, which must be regenerated from the point of the lesion.

²⁵ Lond. Lancet, 1881, p. 495.

²⁶ Casuistische Beiträge zur Lehre von der Neuritis, besonders der Neuritis traumatica und migrans. Inaug. Dissert., Halle, 1886.

²⁷ Handbook of Electrotherapeutics, 1883, p. 86.

²⁸ Berlin. klin. Woch., No. 9, 1877.

Erlenmeyer²⁹ takes exception to these views, maintaining that the time of regeneration depends upon the degree of the paralysis and the amount of the structural changes, as deduced from the character of the electrical reaction by which such conditions are determined. In support of this assertion he cites the first case of M. Bernhardt,³⁰ where recovery occurred first in the biceps and brachialis anticus, next in the supinator, later in the deltoid, and last in the supraspinatus and infraspinatus. The correctness of the views of Erb and Erlenmeyer is clearly demonstrable in our case. The biceps group, presenting *partial De R.*, recovered first, while the supinator longus and the extensors of the forearm, exhibiting *complete De R.*, were the last to show signs of improvement.

The pathology of peripheral neuritis is so well known, that I have purposely refrained from its discussion.

Whether the inflammatory process in this case was of an interstitial or parenchymatous character, or both combined, is of little or no practical importance.

The time required for the regeneration of the nerve-fibres is necessarily influenced by the character and severity of the lesion, by the recuperative powers of the individual, and occasionally by treatment. Recovery sometimes occurs spontaneously, *i. e.*, without the aid of therapeutic measures. In the present case it can be safely assumed that, after the acute symptoms had subsided, the patient neglected all further treatment.

636 LEXINGTON AVENUE.

²⁹ Corresp.-Bl. d. Schweizer Aerzte, 1882, V., 12, p. 619

³⁰ Zeitsch. für klin. Med., Bd. IV., 1882, p. 415.

HEMORRHAGE INTO THE CEREBELLUM AND FOURTH VENTRICLE, AND BY EXTENSION INTO THE THIRD AND LATERAL VENTRICLES.¹

By CHARLES K. MILLS, M. D.

J. C., aged 53, was admitted to the Nervous Wards of the Philadelphia Hospital about July 15th. He was suffering from insular sclerosis and chronic parenchymatous nephritis. He had tremors in both hands, more marked on the left; tremors of the tongue and twitching of the facial muscles, also some loss of power in both arms, more marked on the left side, and this extremity was also slightly atrophied. His left leg was weak, but he had no difficulty in walking. The only intention, however, at present, is to report the hemorrhage from which he died. The patient worked in the diet kitchen and was feeling well until two days before his death, when he began to feel dizzy and complained of headache. He was perfectly conscious, but his symptom grew gradually worse, and in an hour he was completely unconscious. His face became cyanotic. His pulse was 72, and full. He had complete paralysis of the left side of the body, and on the chest on the left side fine muscular twitchings were observed. The pupils were slightly contracted and immobile. He had no convulsions. His temperature at the time of his seizure (one o'clock) was 96°; at two o'clock it was 98.2°; at fifteen minutes after—the time of his death, it was 98.2°. Before death he was very cyanotic and evidently died of respiratory failure. The respirations ceasing a considerable time before the pulse.

Autopsy.—The scalp was much congested with venous blood. The longitudinal sinus contained no clots. The

¹ Presented to the Philadelphia Pathological Society.

dura mater was normal. No evidence of meningeal hemorrhage was present, but under the pia mater covering the posterior and inferior portion of the cerebellum was observed a slight extravasation of blood. The vessels at the base presented a few atheromatous patches. On opening the lateral ventricles a clot was found on the left side, situated in the anterior portion of the ventricle, but not involving the brain substance. The fourth ventricle was filled with black, tarry blood. The main damage to brain tissue was found in the pons, crura, and cerebellum. The tissue in these tracts was plowed up and disorganized. The lenticular nuclei and capsules of the brain were not involved. The heart was hypertrophied, but there were no valvular lesions. The kidneys showed evidences of chronic parenchymatous nephritis. The spleen was enlarged, the liver adherent to the diaphragm and very friable.

Periscope.

EXCERPTS FROM SWEDISH, DANISH, NORWEGIAN AND FINNISH JOURNALS.

By FREDERICK PETERSON, M.D.

HYPNOTISM IN SCANDINAVIA.

L. Bentzon describes a few cases of hypnotization employed as a therapeutic measure ("Ugeskr. f. Læger," R. 4, Bd. 16, S. 579). One was a young hysterical woman, who had suffered for a long period from cephalalgia and pain in the arms, from which she was freed in one séance. Another was a woman, aged 63, who had endured for two years severe rheumatic pains. She was cured in twelve séances.

G. Lütken ("Ugeskr. f. Læger," R. 4, Bd. 16, S. 617) describes 13 cases treated by hypnotism as follows: (1) A man with hypochondriasis was cured by suggestions made in nine séances. (2) A woman with neuralgia of the lingual and œsophageal nerves recovered. (3) A woman with "morbus mentalis" got well after three séances. (4) Another hypochondriac cured after three séances. (5) Chorea in a ten-year-old girl ceased after five hypnotizations. (6) A man was cured of stammering after ten suggestions. (7) A girl of eleven, with chorea and onanism, recovered after fifteen séances. (8) A woman with neurasthenia and neuralgia recovered. (9) A man much improved in mental state for a time, but grew worse subsequently, as the case proved to be paresis. (10) A case of hysteria, with enuresis and pain in the legs, entirely cured after sixteen séances. (11) A woman of 41, with melancholia, who had twice attempted suicide, recovered after six séances. (12) Another case of stammering, in a young man, cured. (13) Nervous condition following an apoplectic attack markedly improved under hypnotization.

S. Hytten ("Ugeskr. f. Læger," R. 4, Bd. 16, S. 648) reports the use of hypnotism in a number of cases of nervous disorder. (1) Cephalalgia of many years' duration, in a twenty-seven-year-old woman, was made to cease for some months by suggestion in a single séance. (2) Hysterical paralysis in a fourteen-year-old girl was cured in a few séances. (3) A thirteen-year-old girl, "shy, tired of home and evil-tempered," was improved in six séances. (4) Hysteria in a sixteen-year-old girl was cured. (5) Sciatica in

a twenty-six-year-old woman disappeared after four séances; was replaced by supraorbital neuralgia, which quickly vanished under suggestion. (6) Cephalalgia and rachialgia cured speedily. (7) Hysterical hemiplegia recovered after three séances. (8) Sciatica cured in another case in five séances. (9) Stammering in a young man easily removed. (10) A tooth was extracted without pain in a woman under suggestion.

F. Björnström has written a 222-page book (recently translated into English) giving a good *résumé* of the development and present standing of hypnotism in the scientific world, intended for physicians and jurists, but also to warn the public in general against its misuse. The author is thoroughly familiar with the work of the Paris and Nancy schools.

O. G. Wetterstrand ("Hygeia," 1888, S. 288, 130, 171) reports a large number of patients suffering from various disorders improved or cured by suggestive therapy.

P. D. Koch ("Ugeskr. f. Læger, R. 4, Bd. 17, S. 10) shows that hypnotism will soon take its proper place in medical science as a therapeutical measure of great importance. Magnus Huss, of Stockholm, has written an 82-page book upon the dangers of hypnotism. While he recognizes its value as a means of treating numerous disorders, he brings forward facts to show that its use may in some cases lead to disturbance of reason, and advises that public exhibitions of hypnotic experiments should be forbidden, and that physicians only should be allowed to practice it, but under certain legal conditions, in order to prevent its use for criminal purposes.

EXTIRPATION OF A CEPHALOCELE.

Dr. C. A. Bergh ("Nor. Med. Archiv.," 2d quarter, 1888) describes the case of a girl baby, five weeks old, admitted into the Gefle Hospital in 1886, with an occipital hydrocephalocele. It was an elastic pedunculated tumor, 10 cm. in diameter, springing from the posterior fontanelle. It increased in size on crying. Pressure revealed tissue of firmer consistence in its interior, but caused no cerebral symptoms. A clamp was applied, and the tumor punctured and removed under antiseptic precautions. A piece of brain the size of a hazel nut was evacuated with the serous fluid. The clamp remained in situ for twenty days. Complete recovery took place, but there is at present a slight sign of return.

ON THE DIRECT HEREDITY OF INSANITY.

In the ("Hosp.-Tidende," R. 3, Bd. 5, S. 1129, 1172, 1190, 1220) Dr. Th. Eibe presents some interesting statistics with reference to this subject from the Nørrejske (Denmark) asylum. Out of 3,500 patients treated here since the opening of the institution, there were 55 who had children later admitted insane. There were 19 fathers with 8 daughters and 15 sons, and 36 mothers with 15 daughters and 24 sons—or in all 55 ascendants and 62 descendants.

Fifty-four of these ascendants had altogether 294 children. Of these children 52 died in early childhood, 91 are or have been insane, 1 is epileptic; and of 150 nothing pathological is known. With regard to the offspring of the descendants, the time is not yet ripe for exact statistics, as the figures will change in a few years. However, 37 per cent. of the descendants are entirely childless, 35.5 per cent. have had children, and in 27.4 per cent. the possibility of having children cannot yet be excluded. The following table gives some information as regards prognosis:

<i>Form of Insanity in Ascendants.</i>	<i>Descendants.</i>		
	<i>Age at Outbreak.</i>	<i>Recovered.</i>	<i>Puberty Psychoses.</i>
Periodic Forms	19.9	11 %	89 %
Melancholia.	21.1	59 "	76 "
Mania	23.2	67 "	67 "

EMOTIONAL DISTURBANCES AS A CAUSE OF DISEASE.

Some cases of interest are cited by Dr. A. Sell ("Hosp.-Tidende," R. 3, Bd. 5, S. 961, 985) in an article upon this topic. Among them are two of apoplexy following mental strain; a case of epilepsy after fright; eclampsia in a four-year-old child due to a slight punishment; a case of renal colic beginning immediately after receiving bad news, and ending with the discharge of a concrement; and one of great increase of bodily temperature after extraction of a tooth.

FOUR CASES OF APHASIA, WITH AUTOPSIES.

In the "Upsala läk. förhandl." (Bd. 21, S. 380-93) S. E. Henschen reports in detail the following cases with illustrations:

CASE I.—Amnesic aphasia and agraphia, and at the beginning aphemia, in a woman aged sixty-two, with stenosis aortæ. She could repeat every word said to her, but spontaneous expression of thought was completely lost. Would use incorrect words. Duration of aphasia eleven days. Death two years later. Autopsy: Softening in a part of F³ and F² and neighboring C⁴.

CASE II.—Word blindness, amnesic aphasia, and in the beginning ataxic aphasia, in a laborer, aged fifty-five. Apoplexy, right hemiplegia. Could see letters, but make no word of them. Understood what was said to him. Could not read or write. Death in about a month. Autopsy: Softening in the left gyrus angularis extending downwards into the depth of the fossa Sylvii.

CASE III.—Word blindness with amnesic aphasia, and partial word deafness, in a woman aged fifty-seven. Apoplexy without paralysis. Autopsy: Softening in the posterior part of the first temporal convolution, extending over the posterior part of the second also and to the gyrus angularis.

CASE IV.—Word blindness, motor aphasia and right hemiplegia in a man aged twenty-nine. Autopsy: Extensive disorganization of left hemisphere about F³, foot of F², lower part of both central convolution, P², a part of P¹, T¹ and T².

HEMIATROPHY OF THE TONGUE OF BULBAR ORIGIN.

S. E. Henschen ("Upsala läk. förhandl.," Bd. 21, S. 347) collects eight known cases and adds one of his own, which is as follows:

At nine years of age the boy had scarlet fever, and at fifteen a fall from a horse. Although the fall did not hurt his head or cause him to lose consciousness, his left arm was weak subsequently, and some contracture of the fingers of the left hand followed. Deformity in the tongue had never been noticed until he entered a hospital for uræmia and pulmonary œdema. There was very marked atrophy of the right half of the tongue. Left abductor paresis of the larynx. Left hand claw-like. No symptom of tabes. Right soft palate atrophied. The author regarded the atrophies as a result of scarlet fever. The nuclei affected were some of those of the hypoglossus, vagus, accessorius and facialis. He would have considered the *main en griffe* a part of the process had not the patient declared this to be the result of trauma.

A CASE OF LANDRY'S PARALYSIS, WITH RECOVERY.

L. Lorentzen describes the following case in the "Ugeskr. f. Læger" (R. 4, Bd. 17, S. 597): A woman, aged thirty-six, after exposure to cold, noticed a paresis in her legs, which grew worse rapidly. Nine days later there was complete paralysis of both legs and almost complete of both arms and of the muscles of the neck. She could make weak movements of the fingers only, and move the head slightly from side to side. Later there was marked paresis of the lips and of the abducens of both eyes, but none of the tongue, palate or pharynx. Respiration, defecation and micturition normal. The face was somewhat congested, temperature raised a little the first five days, pulse accelerated, but never above 92. There was no splenic enlargement, no albuminuria. Fundus oculi normal. The deep reflexes were absent, the skin reflexes very weak. Paralysis flaccid. Reaction of muscles to both currents retained. There was no anæsthesia and no pain or tenderness. Improvement took place gradually, first in the arms, lips and trunk muscles, last in the eye muscles and legs. Complete recovery in three months. There was never any atrophy of the muscles. The author considered the diagnosis to lie between acute poliomyelitis anterior, acute multiple neuritis and Landry's paralysis. He believed the case to belong in the latter category, and localized the pathological process in the spinal cord and medulla oblongata.

RHEUMATIC TIC CONVULSIF WITH THICKENING OF THE TRUNK OF THE FACIAL NERVE.

S. E. Henschen ("Upsala läkarefö. förhandl.," Bd. 23, S. 219) relates the case of a twenty-seven-year-old teacher who had suffered a number of years from tic convulsif on the left side of the face after exposure to cold. The trunk of the facial nerve and some branches of the trigeminus were found to be tender and considerably thickened. After six weeks' massage the swelling and tenderness disappeared, and she was discharged recovered. The author believes that an inflammatory process in the sheath of the facial nerve was produced by cold, and that this was the cause of the convulsif tic. He looks upon migraine seizures as due to thickening in branches of the trigeminus, a perineuritis, and has corroborated this frequently since his first expression of this opinion in 1881.

THE PATHOLOGY OF ACUTE POLIOMYELITIS ANTERIOR.

J. Rissler reports ("Nord. Med. Archiv.," Vol. XX., 4th quarter) the results of his study of the material from five cases. Three died in the first week of the disease, one after seven weeks, and one after eleven years. His investigations corroborate the observations of others and the general opinion held as to the pathological anatomy of the disease. The inflammatory process was rigorously limited to the anterior horns, and the degenerative changes produced in the ganglion cells were very characteristic. The contribution is from the laboratory of the nerve clinic of Prof. Wising in Stockholm, and its being written in German, places the 58-page paper within the reach of those interested. Some remarkably beautiful plates are published with the article.

PSEUDO-HYPERTROPHIC MUSCULAR ATROPHY IN TWO BROTHERS.

J. W. Runeberg and E. A. Homén describe these cases in the proceedings of the "Finnish Medical Society" (Bd. 29, No. 4, S. 212 and 213). Mother feeble-minded, father alcoholic, maternal uncle insane. One boy, aged eight, began at five to exhibit an uncertain and wobbling gait, the legs gradually becoming large, and the upper part of the body emaciating. Upon his entrance to the clinic, the muscles everywhere, except on the legs, flaccid and atrophied; very great lardosis; thigh and calf muscles very large and prominent and firm in consistence; intelligence feebly developed.

The other boy, aged fourteen, also began at about the age of five to present unevenness of gait. He grew rapidly worse, so that for two years past he has been confined to bed. On entering the hospital he could not raise himself in bed. Muscles of upper part of body greatly atrophied, and calves only are voluminous and firm. The reaction of muscles to galvanic and faradic currents greatly diminished. Feeble minded.

TREATMENT OF PERIODICAL DRUNKENNESS WITH STRYCHNINE SALTS.

Max Buch gives an account of the successful experiences of himself and others in the cure of inebriety by this means ("Finska läk. handl.," Bd. 29, No. 4, S. 204). He follows Popoff, Tolwinsky, Parzewsky, and Sawatyky in undertaking this method. Either the sulphate or nitrate of strychnine may be employed, in solution or pills, or by hypodermic injection. The doses used are .001-.003, once to thrice daily.

EXCERPTS FROM ITALIAN JOURNALS.

By GRACE PECKHAM, M. D.

RESULTS OBTAINED BY SUSPENSION IN CASES OF TABES.

("Rivista Sperimentale di Freniatria e di Medicina Legale," 1889.)

Suspension was practised a number of times in eleven cases, with the result that, with the exception of two cases, there was an improvement in walking, but no alleviation of the pains. The results of the experiments neither contradicted nor affirmed those of Salpêtrière.

CONCERNING MOTOR HALLUCINATION.

(Notes by Prof. A. Tamburini, "Rivista Sperimentale di Freniatria e di Medicina Legale," vol. xv., fasc. iv., p. 444.)

The writer reports the case of a peasant of low intelligence, twenty-seven years old, who at fifteen, after a grave affection, had an attack of melancholy, in which she had a hallucination of hearing a voice which said to her, "Lost forever," which lasted about four months. In March, 1888, after she had been married a short time, she had a return of melancholia, was apathetic and sleepless; she had also some dysmenorrhœa and leucorrhœa. She said that "words formed themselves in her mouth." When she attempted to take food, she felt come into her mouth the words, "Thou couldst eat a serpent," "Thou couldst swallow a live toad." When praying there would be maledictions against divinity and obscene expressions. Later she was obliged to repeat in a high voice the words which formed themselves continually in her mouth, which she must repeat in a rapid manner. She also had clonic spasms. There was a light rhythmical movement in the tongue and in its tip, accompanied by a sound which could be heard when her mouth was closed. Her lips were motionless. There were also clonic spasms in single groups of muscular fibres, especially those used in articulation.

The psychical examination was imperfect, owing to the limited intelligence of the patient. The physical examination showed that the cranio-facial type tended to the cutinoid; forehead low; nose flat; intermittent asymmetry of the pupil; oscillatory movement of various muscles of the face; sensibility normal, and symmetrical for both sides of the body; organic functions normal.

The author, after reviewing the case and the literature of the subject, including the various theories, comes to the following conclusions:

1. That besides the purely sensory hallucinations, it is of interest to distinguish the motor hallucination, which displays itself more especially in the movements relating to speech, but may also show itself in any part of the body capable of movement.

2. That the seat of this last kind of hallucination ought to be located in the cerebral cortex.

3. That according to the degree of irritation of these same centres, there would be simple hallucination of movement or the transformation of this into an inconvertible impulse, even to a relative convulsion.

4. That, moreover, taking specially into account the physiological and clinical data, in which is admitted the mixed sensory-motor nature of all the cortical centres, there is, in every hallucination a part proportioned to that belonging to each centre, so much of the sensory image as is appropriate to the motor.

Miscellany.

THE SOLVENT PROPERTIES OF BUFFALO LITHIA WATER.

The "Virginia Medical Monthly," Dec., 1889, contains a paper by Dr. John Herbert Claiborne, of Petersburg, Va., in which an account is given of a case of severe lithiasis treated effectually by large doses of lithia water. The patient had a violent attack of nephritic colic in August last, passing gravel from the kidney into the bladder, where it remained several weeks, setting up severe inflammation and causing distressing symptoms, and finally forming a calculus. Crushing the stone or performing lithotomy was considered inexpedient on account of the intense inflammation. The patient was put to bed, the diet restricted to milk, and opium suppositories administered in sufficient doses to relieve tenesmus and pain. The patient drank from half a gallon to a gallon of Buffalo lithia water, and in about ten days commenced to pass in surprising quantities what appeared to be the detritus of the gravel. Urine that had been deposited on a clean board, left, in evaporating, about a drachm or two of sediment like whitewash, containing phosphates, urates, etc. This continued for a week. Then the bladder was washed out with a warm solution of boracic acid, and the patient, at the time of writing, reported himself well and free for the first time from all kidney or

bladder trouble. An editorial in the same journal speaks of the attention Dr. Claiborne's paper must necessarily attract, on account of his reputation as a close observer and a cautious writer.

Prof. George B. Fowler, in the "Reference Handbook of Medical Sciences" (vol. i., p. 718), pronounces himself as favoring the use of this lithia water in lithiasis, etc. So also does Dr. H. B. Millard, in his standard "Treatise on Bright's Disease." The Virginia waters are more than respectable rivals of the celebrated Vichy Spring. L. F. B.

THE JOHNS HOPKINS HOSPITAL BULLETIN.

The trustees of the Johns Hopkins Hospital have authorized the issue of a monthly publication with the above title, to contain announcements of courses of lectures, programmes of clinical and pathological study, details of hospital and dispensary practice, abstracts of papers read and other proceedings of the Medical Society of the hospital, and all other matters of general interest in connection with the hospital work. Nine numbers will be issued annually.

The first—December, 1889—is at hand, and contains readable and instructive papers on the following subjects: A Brief Account of the Johns Hopkins Hospital; Opening of the Nurses' Home and Training-School; Preliminary Report of Investigations concerning the Causation of Hog-Cholera, by William H. Welch, M.D.; On the Value of Laveran's Organisms in the Diagnosis of Malaria, by William Osler, M.D.

Among various attractive announcements, the following is of especial interest to students of psychiatry: "A course of five didactic lectures for graduates in medicine will be given by Dr. Hurd, superintendent of the hospital and professor of psychiatry, Johns Hopkins University, during January and February, upon the Genesis of Delusions, the Insanities of Childhood, Pubescent, Adolescent, Climateric, and Senile Insanities, Insanities from Constitutional Disease, and Clinical Groupings of Insanity." L. F. B.

NOTE UPON THE PAPER, "THE RELATION OF THE THALAMUS," ETC., IN THE NUMBER FOR JULY, 1889.

Owing to the failure of proof to reach the author, certain corrections and additions should be made.

Page 436. After the title insert: Presented at the Washington Meeting of the American Neurological Association, August, 1888.

Page 437, about middle, for *striato-thalamic groove*, read *tenial sulcus* ; for *plexal groove*, read *fimbrial sulcus*.

Page 438, 8th line from top, for *Mihalkorics*, read *Mihalkovics* ; 18th line, for *Gegenbauer*, read *Gegenbaur* ; 20th line, for *Vicgdazyr* read *Vicq d'Azyr* ; 9th line from bottom, after *deny* insert *that* ; 4th line from bottom, for *or* read *of* ; 3d line from bottom, the semicolon should be replaced by a colon and dash, :—.

P. 440, 2d line, for *mammels* read *mammals*.

P. 441, 9th line from top, *dele* the comma after *and* ; 2d line, in place of *my paper 56*, read *the N. Y. Medical Journal, April 26, 1884* ; 10th line from top, for *all*, read *most* ; 11th line from top, *dele* the words from *mammals* to close of sentence.

P. 442, 13th line from bottom, insert a comma after *paracæles* ; 8th line from bottom, omit *that* ; 12th line from bottom, for *all*, read *most* ; 11th line from bottom, *dele* the words, *excepting the anthropoids*.

P. 443, 7th line from bottom, for *diocælian* read *diacælian* ; 5th from bottom, for *lateral* read *laterad*.

A corrected reprint will be sent upon application to the author. At the meeting of the Association of American Anatomists, in Philadelphia, Dec. 26, 1889, the subject was considered with special reference to the condition of the parts in the anthropoid apes, and the term *paratela* proposed for the fusiform zone of membranous parietes that, in the human adult, intervenes between the divaricated fimbria and tenia and is sometimes attached closely to the dorsal surface of the thalamus ; Schwalbe calls it *lamina chorioidea lateralis*. Figures such as are suggested on p. 443 are given in the last volume of Wood's "Reference Handbook of the Medical Sciences," Figs. 4750, 4751.

B. G. WILDER.

Society Reports.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, October 28, 1889.

The Vice-President, Dr. WHARTON SINKLER, in the Chair.

A CASE OF HEMIANOPSIA WITH DYSLEXIA; ALSO JACKSONIAN EPILEPSY.

By CHAS. K. MILLS, M.D., and G. E. DE SCHWEINITZ, M.D.

J. H. C., aged forty-five, white, collector, was sent to Dr. Mills by Dr. J. H. Packard, of Philadelphia. He had no history of syphilis, but about six years ago had two attacks of acute rheumatism. He had suffered with headache since boyhood. Some failing sight and dizziness had come on during the summer of 1888. He has headache at time, but not steadily; sometimes he goes to bed with it and gets up without it.

December 17, 1888, while walking in the street, his left hand and arm felt queer, and became affected with spasm, shaking so that he had to hold it with the other hand. He got into a car, the arm twitching and jerking, and before he reached home had convulsions with unconsciousness. He was taken home, and found, when he came to his senses, that he was partially paralyzed in the right arm and leg, the loss of power being most marked in the leg. He thinks he has no paralysis of the face, but his speech was much affected for two or three weeks. He soon recovered from the paresis, but had a second "fit" in about three months, having three spasms within an hour. He does not know how these spasms began, how they spread, and how long they continued. Since then he has had no seizures, and this series of attacks did not leave him paralyzed or his speech affected; but he discovered that he could not see half of objects to his right.

He reads in a very peculiar way—slowly, and pronouncing each word separately, or at the most two or three words; he seems to have difficulty in seeing the word; he *says* that he sees it plainly, but that he soon gets mixed and confused. Although he sees the words, it takes him a long time to form the idea of them. Sometimes

he can scarcely make out words. In his own way he can go on for a short time reading, and then his brain seems to exhaust; he gets confused. Occasionally, when walking in the street, he imagines he sees something that does not exist—always to the right. Hearing and touch are normal.

The following notes, by Dr. de Schweintz, describe his ocular condition:

In the right eye the sharpness of sight was equivalent to $\frac{2}{7}$ of normal; in the left eye, $\frac{2}{5}$ of normal. This deficiency in visual acuity was probably due to the presence of mixed astigmatism. In the right eye the optic disc was a vertical oval, bounded at its outer margin by a black line, its surface a little woolly, and all the capillaries injected; the edges of the disc, however, were not obscured. In the left eye the disc was distinctly grayer in color, its hue being manifest through a superficial injection of the surface capillaries. The temporal half of the disc was unobscured; the nasal edges slightly blurred. In neither eye was the disc swollen, nor were there any splotches nor hæmorrhages in the retina. The pupils of both eyes were equal in size, and reacted normally to the changes of light and shade, convergence and accommodation. The hemiopic pupillary inaction (Wernicke's symptom) was not present in either eye. There was complete right lateral hemianopsia, the field of the left side being proportionately much smaller than its fellow on the right, and both the preserved fields exhibiting concentric contraction. The dividing-line on the left side almost cut the fixing point. That on the right side, on the horizontal meridian, touched the fixing point, while above and below this it spread five degrees from the centre, making a curious reëntering angle at this point.

Dr. MILLS said: This seems to be an important case in the study of localization. It presents four or five important features: (1) The Jacksonian spasm confined chiefly to the left hand and arm, in the first attack at least beginning and continuing for some time before consciousness was lost. (2) Temporary paresis of the arm and leg upon this side. (3) Right homonymous lateral hemianopsia. (4) Absence of Wernicke's symptom. (5) Dyslexia. The hemianopsia seems without doubt to be cerebral in character, Wernicke's symptom and choked discs being absent. The lesion is back of the primary optic centres, and is therefore either in the optic radiations, the cuneus, or the lateral convexity of the occipital lobes. As between these the lesion is probably in part in the optic radiations. The optic radiations coming

from the primary optic centres curve around the posterior horn to the occipital convolutions. In the acts of reading and writing there is of course a conveyance of impressions by a commissural channel between the visual areas of the cortex and the arm and leg centres. We are greatly lacking in a knowledge of the coarse anatomical relations between these commissural fibres, the horns of the ventricles, the lateral ventricles, and the cortex. There seems to be a position where the commissural fibres intermingle with or abut against the optic radiations. This may be perhaps where the parietal, temporal and occipital lobes come together around the roof of the ventricle. The lesion is in the centrum ovale at a point near what might be called the parieto-occipito-temporal junction. It is well back over the roof of the ventricle, perhaps above the place where the lateral ventricle joins the posterior horn.

Dr. DE SCHWEINITZ said that he went further than the mere ocular examination, and looked into the manner of reading and writing. This man could read two or three words and then threw away the paper with a look of disgust. Again he would pick up the paper, and, after reading a word or two, throw it down. The same thing occurred when he attempted to write. He wrote a few words, and then his hand failed to inscribe further letters, while his face gave evidence of chagrin at the abortive attempt. The points in the ocular examination were absence of optic neuritis, the presence of right lateral hemianopsia, unassisted with Wernicke's pupillary reaction. He did not wish to be understood as saying there was no disease of the optic nerves, but there was no coarse lesion like choked discs—that the nerves were gray, but not in a state of neuritis. The optic nerves were certainly not healthy, or the preserved fields would not be contracted.

In the cases reported by Berlin, six in number, and one by Niden, post-mortems were made in several, and the lesion was found in the white matter near Broca's convolution. I have not at hand the original paper, but the following *résumé* appears in Swanzy's "Handbook of Ophthalmology:" "Dyslexia consists in a want of power on the patient's part to read more than a few—four or five—words consecutively, either aloud or to himself. * * * Although in most cases the dyslexia disappeared in the course of a few weeks, yet other symptoms soon followed its first onset, such as headache, giddiness, aphasia, hemianopsia, paralysis of the tongue, hemianæsthesia, hemiplegia," etc.

Dr. H. C. WOOD: The objection to this theory is that

one of the most important symptoms is omitted in making up the theory, that is, the contraction of the fields of vision. I do not see how it is possible to have lateral hemianopsia with contraction of the field, with the optic nerves healthy and yet not have a lesion of the cuneus or the cuneal region of both sides.

There is one point that we forget in the discussion of these cases, and that is, that we do not know whether the symptoms presented coexisted in the beginning, or whether some are primary and some secondary. Dr. de Schweinitz will recall a case of tumor of the temporal lobe, which we studied together, in which we had all the symptoms described to-night but the dyslexia. If we suppose a tumor a little more forward and higher up, encroaching upon the Broca region and interfering with the posterior cerebral artery, and considered that the hemianopsia is due to optic neuritis, we will have a possible explanation of the present case.

EXHIBITION OF A CHINESE BRAIN.

By Dr. JAMES HENDRIE LLOYD.

The study of the comparative morphology of the brains of the various human races is of more interest to the anatomist and evolutionist than to the clinical neurologist or the practical alienist. I present this brain to demonstrate the truth of this statement. It does not exhibit any very marked differences from brains of the Caucasian and other races. The description of such brains pertains entirely to pure and abstract science, and I think is of very little practical importance.

From the way in which Chinese brains have sometimes been regarded, the impression seems to be that the Chinese are an inferior race and that in them we have to look for evidences of low type. I think that is illogical and without foundation of fact or historic evidences. The Chinese are not a low race of men in any sense. The Chinese stock is different, historically and ethnologically, from the Caucasian stock. It has gone on in its own line of development, and may have produced some slight differences in the architecture of the brain which might appeal to the eye of those expert in examining the morphology of the brain; but such differences are not necessarily evidences of inferiority.

We must remember that there are different kinds of Chinese, just as there are different kinds of Americans.

I have no doubt that some Chinese have worse-developed brains than others. This brain was removed from such a Chinaman as we frequently see in this country, a poor, miserably developed little Mongolian, who died of tubercular meningitis, under my care, in the Philadelphia Hospital.

There is no doubt that in this particular Chinese brain there is a certain simplicity of structure and absence of richness of convolutional development. In other words, the brain looks almost like some of the schematic drawings seen in books exhibiting the primary fissures. There is also in the occipito-parietal fissure what has been shown to be of rather a Simian type, that is, the external *pli du passage*, or bridging convolution, which in a well-developed brain comes to the surface, forming a well-rounded convolution, is in this hemisphere quite distinctly below the surface. This is perhaps the strongest evidence of this brain being below ordinary development. Besides this there is nothing to warrant the assertion that this is a low-type brain.

General inspection of the surface of both hemispheres shows a simplicity in the arrangement of the fissures and convolutions. There are some slight differences between the two hemispheres; for instance, the right Sylvian fissure is much shorter than the left. Another notable feature (proving perhaps a rather unusual tendency to a confluence of fissures) is the fact that the right first temporal fissure is confluent with the anterior portion of the inter-parietal; hence the *angular gyrus* (about which as a centre of vision so much was formerly written) is not well marked in the right hemisphere. Any other differences which I have noted in this brain are entirely of minor importance, except perhaps the following:

In the right hemisphere, as already stated, the superior external *pli de passage* (the convolution uniting the parietal with the occipital lobe) is small, narrow, and distinctly depressed below the surface; while in the left hemisphere it is well developed and up to the surface. The imperfect development of this convolution is regarded by some as an evidence of low type. When below the surface, it permits the division of the parietal from the occipital lobe to be much more clearly seen than is usual in the more highly developed human brain. It is a condition, I believe, not uncommon in the Simian brain.

Dr. CHAS. K. MILLS.—While these studies of brains may at present be useless from a medico-legal standpoint, yet

the fact is that in low-type brains, whether Chinese or Caucasian, there is a great simplicity of convolutions and fissures with certain special peculiarities. This is also seen in criminal and imbecile brains. These facts show that this is fundamentally a correct method of study. It is, however, not the only method, and is to be used in connection with other methods, as microscopical and pathological. In order to reach positive conclusions, we must study a large number of brains, both from the lower classes and from the higher classes.

Dr. J. MADISON TAYLOR said that in forming an opinion as to facial peculiarities from examination of the brain, and especially among the older Oriental peoples, it is needful to take into careful consideration class distinctions. These among the Chinese are drawn into the sharpest lines. Development of special types along these lines, running as they do for centuries, is inevitable. How this may be manifested by brain-shapes we can only learn by comparing many of one class with many of another. So far we have only the lowest offscouring among us of the Chinese representatives of the droppings of all classes. Among these we may look with small hope for aught but low types. On the Pacific slope a few of the best upper classes—the artisans and merchants—are seen. Here can only be had the brains of the pariahs, and even these bred in sin till the resultant is obviously unlovely.

Dr. JAMES HENDRIE LLOYD.—As I have said, these studies are of importance to the comparative anatomist and the evolutionist, but they are of little clinical interest, of no practical value to the alienist, and devoid of medico-legal importance. It is nothing but an assumption to say that these studies are made upon a low-type race. I do not think, because there are social castes, that the lowest caste has a different type of brain from the others. Caste is a matter entirely of social development, and has nothing to do with such an occult matter as the size and shape of the cerebral convolutions. It is notorious that examples of idiocy and low-type brains are common among the higher classes in countries in which class distinctions are most recognized, and, on the other hand, that the best-developed brains often spring from the so-called common people. The infusion of the blood of the people is all that saves many privileged classes from sinking into a very low type indeed.

Stated Meeting, November 25, 1889.

Vice-President, Dr. WHARTON SINKLER, in the Chair.

Two recent specimens were presented by Dr. CHARLES K. MILLS :

I.—HÆMORRHAGE BENEATH THE SCALP, AND CEREBRAL AND CEREBELLAR HÆMORRHAGES IN A CASE OF ALLEGED TRAUMATISM.

This specimen was from a woman about sixty years of age, of whom it was alleged that she was struck on the head by her husband, both she and her husband being at the time drunk.

The following are the notes taken of the condition of the patient after admission to the hospital :

She was admitted on November 22d, at 10.30, in an unconscious condition. She lies upon her back, eyes closed ; perfectly quiet, and does not move unless disturbed. On examination an area of discoloration is found on the left cheek, extending to the eye on the same side. The left eye is swollen and blackened, and the whole of the right side of the face seems slightly swollen. On the right side of her head, about the posterior superior angle of the parietal bone, is an area about two inches in diameter, nearly circular in form, which feels like a boggy mass underneath the skin. No fracture of the skull is apparent. Both ocular conjunctivæ are chemosed, especially on the right. Her head deviates to the left side, and the left eye is turned to the left and moderately dilated ; the pupil is immobile. The right eye is straight, the pupil is very small and immobile. She has no marked paralysis of the face ; the left cheek is puffed, and is drawn in and out on expiration and inspiration. The left arm is paretic—not completely paralyzed ; she seems to have slight power of moving this arm, but only does so when irritated. The forearm is always carried at a right angle with the arm ; the wrist and fingers are partially flexed. The right arm is considerably more rigid than the left ; the forearm is at an obtuse angle to the arm ; the wrist and fingers nearly extended. Both hands are cold. The left lower extremity is fully extended, and the right leg is partially flexed at the knee ; the foot is extended and inverted, and partial loss of power is undoubtedly present in both extremities. The feet are cold. The left knee-jerk is diminished, the right normal. Sensation

is present in the face, body, and lower extremities, but seems to be abolished in the upper extremities. Her breathing is stertorous and at times of the Cheyne-Stokes type. She swallows with difficulty, and passes her feces and urine involuntarily. Her pulse is regular, of moderate volume. The heart and lungs appear to be normal. Examination of the urine gives negative results.

The following is a record of temperature, pulse, and respiration :

Temperature, 97°;	pulse, 78;	respiration, 21—	on admission.
" 97.2°;	" 72;	" 24—	1 A. M.
" 97.4°;	" 64;	" 24—	4 P. M.
" 97.1°;	" 90;	" 18—	noon.
" 98.4°;	" 100;	" 24—	7 P. M.
" 99.1°;	" 64;	" 20—	10.30 P. M.

November 24, 1889.—This morning the patient seemed to be rather improved; her breathing was less stertorous in character, more regular, and her pulse quite good. She is still unconscious. Motor and sensory disturbance almost the same as yesterday, except that sensation seems to be present in the upper extremities but abolished in the lower. When pressure or percussion is made over the injury to the head the patient moves her arm on the right; this may be due to pain. The left pupil is not quite so much dilated, and the right one seems a trifle larger; both are immobile.

November 24, 1889.—In the evening she relapsed into complete unconsciousness, her breathing became more labored, her pulse rapid and weak; there were trachial rales, and the patient died at 5.30 P. M.

Temperature, 98.4°;	pulse, 85;	respiration, 19—	1.30 A. M.
" 98.4°;	" 76;	" 18—	4.30 A. M.
" 98.2°;	" 80;	" 22—	8.30 A. M.
" 98.2°;	" 100;	" 22—	11.30 A. M.
" 99.6°;	" 104;	" 20—	1.30 P. M.

Autopsy.—The scalp over about one-half the parietal bone and the upper border of the squamous portion of the temporal bone was infiltrated with blood having a bruised appearance. On separating the scalp from the skull a flattened clot was found in the middle of this area between the scalp and bone. On removing the skull-cap no clot was found between the dura mater and the bone. There was a small subdural ecchymosis, subdural related to the

middle of the squamous portion of the right temporal bone. A clot five inches long and two and one-quarter inches broad was found underneath the dura mater covering the two upper convolutions of the temporal lobe, and reaching over the lower border of the parietal and frontal lobes. Another clot, one inch by one and one-half inches, was found on the surface of the left cerebellar hemisphere at its anterior and lower border. No blood was found at the central portion of the base of the brain. On opening the horns of the lateral ventricles and the ventricles themselves, no hæmorrhage was found. Sections through the ganglia and tracts showed that the large hæmorrhage originated within the cerebrum, probably from a point along the external border of the lenticular nucleus.

II.—EXTENSIVE CORTICAL SOFTENING.

This specimen was removed from a man who in 1884 was in the wards of the Philadelphia Hospital, but left and found his way back into the out-wards, where a few days ago he was attacked with apoplexy. He had a small right leg—an old atrophic paralysis; it was two or three inches shorter than the left leg, and much smaller in all its measurements.

The arms are apparently of the same size; if there is any difference, the right is larger than the left, as is found in right-handed men.

His condition on November 21, 1888, at three P. M., was noted as follows: He lies upon his back with his head turned toward the left; opens and closes his eyes from time to time, and moves them about in different directions. His breathing seems somewhat labored, with some tendency to ascent and descent, but no true hiatus. Nothing can be determined from the man himself, as he is not conscious to respond to questions or to perform any willed movements. He has a tremulous movement of the extremities. It is difficult to obtain any definite results as to sensation. Sharp points are evidently felt on the left side of the face. He does not show evidences of sensation on either side of the trunk or the extremities; when tested by a pin, the results are unsatisfactory. His right arm is contracted at an angle of a little more than 45° over his abdomen. His fingers are turned inward, slightly clawed; the middle, ring, and little finger are more firmly flexed than the index finger or the thumb—the index finger and thumb being brought together in nearly a pen-holding position. He has little or no power in the right arm, but does occasionally use it,

although he cannot fully extend it nor use the fingers and hand.

His right foot and leg have a purplish appearance—toes are all clawed and bunched together; the nails are very deficient. The whole foot is in a bad vaso-motor or trophic condition. The leg does not seem cold. He does not move the right leg—the palsied and atrophied one—but he frequently moves the left arm and leg, and the latter is quite spastic at the knee.

Knee-jerk is abolished, both on the right and left sides. His condition as to motor power seems to be that he has an old palsied and atrophied lower right extremity, and is at present suffering from an apoplectic attack, which has affected his right face and arm, and has probably deepened the paralysis of his right leg, if this was not already complete.

The electrical reactions to faradism, November 22, 1889: The muscles of the quadriceps extensor on the right side respond well, but other muscles of the thigh do not. There is no response in the right leg to the strongest currents. At the plantar surface of the foot a medium current produces contraction of the flexor brevis digitorum, and flexor brevis pollicis; the others do not respond. His urine passes involuntarily.

The pupils are equal, with a median degree of dilatation. The wrinkles on the left forehead are much more defined than those upon the right side; orbicularis palpebrarum of the left side is much stronger than on the right. The right side of the face and mouth droop somewhat.

This man has the peculiar state of consciousness noticed in some cases of apoplexy, namely, while irresponsive to questions and tests, he opens or half opens his eyes and looks about, as if half conscious of his surroundings. He does not speak, nor does he seem to know what is said to him.

The following notes were taken November 22, 1889: There is a decided deviation of both head and eyes to the left. The pupils remain about the same as before. The sensory and motor symptoms are unchanged, except that the spasticity of the muscles of the left thigh is increased, producing flexions of the leg at an obtuse angle to the thigh. There is slight spastic contraction of the flexor muscles of the left arm, but not nearly so well marked as that of the thigh on the same side. He moves his left arm at intervals, but no voluntary movements of his left leg are noticed. The whole of the right side is immobile. On the

next day there are no changes in the symptoms except that all are more marked and the patient is failing rapidly. He died at 3.45 P. M.

Autopsy.—November 23, 1889. The upper extremities of the right ascending parietal and frontal, and right superior frontal convolutions, seem to dwindle in size as compared with the corresponding convolutions on the opposite side; large diffused area of cortical softening, involving about one inch of the ascending parietal, lower border of the superior parietal, and entire inferior parietal convolutions on its lateral aspect, partly on its Sylvian aspect. One of the branches of the cerebral vessels was occluded, and the middle cerebral itself, where it gives off its four cortical branches, is partly occluded. Loose clots are found in several portions of the middle cerebral region. There are no lesions of the interior of the cerebrum. The cord was removed and will be subjected to microscopical examination. The kidneys show considerable interstitial changes.

TUMOR OF THE BRAIN (EXHIBITED BY DR. GUY HINSDALE).

The patient from whom this tumor was taken was under the care of Dr. R. M. Girvin, of West Philadelphia. He was seen by Drs. W. W. Keen, Morris J. Lewis and C. A. Oliver. As Dr. Keen will report the case in full elsewhere, I will state only the main facts relating to it.

CASE OF N. F., æt. 17.—In November, 1886, he fell from the roof of a stable twenty feet, breaking his right thigh and left forearm. He also struck and cut his chin, knocking out one of his front teeth. His head was not known to have been struck. He was rendered unconscious by the fall for a considerable time, but by the next day all the cerebral symptoms had passed away. Consumption, diabetes and insanity exist in three persons in the two prior generations of his family. His father, his sister and himself also have been subject to asthma, and he to chronic nasal catarrh.

In April, 1889, his right knee sometimes gave way under him, and gradually the right arm became affected to a less degree, and, as throughout the whole case, so in these two limbs, a curious alteration took place, the leg sometimes being weak without the arm being attacked, and *vice versa*. Headache began about this time with a marked dragging

gait, and sometimes nausea and vomiting. Soon afterward two convulsive attacks occurred with blindness.

Dr. Keen saw him first on October 1, 1889. The evidences of tumor were very clear, and with it he had ophthalmoplegia, both interna and externa. This ophthalmoplegia varied curiously in degree from day to day. The lower part of the right face was also paralyzed. In view of the extensive symptoms, both of the cortex and the base, it was thought that one very large tumor, or possibly two small ones, existed, and it was decided not to operate. The boy died on November 18th. The post-mortem was made thirty hours after death. The outer surface of the brain looked entirely healthy, but the moment that the median surface was exposed a tumor was found protruding into the median fissure from the left side. In the manipulation necessary to remove the brain from the skull, this tumor enucleated itself. It was $3\frac{3}{4}$ inches by $2\frac{3}{4}$ inches, and lay directly under the motor area; it lay in the white substance and extended well down toward the base.

DISCUSSION.

Dr. OLIVER.—As it is the intention of Dr. Keen and myself to present the details of this case in full at a future time, I will merely state that when I first saw the patient there was a marked double neuro-retinitis of equal degree of swelling and density, associated with right homonymous hemianopsia; the remaining fields being limited to light perception, which on the right side had become almost entirely lost. Both pupils were dilated to seven millimetres, and the irides were absolutely immobile to the strongest light stimulus. All of the extra ocular muscles were impaired in both monocular and associated action; the utmost movement of the superior recti were about four millimetres; the inferior about three millimetres; right associated movement about one or two millimetres; left associated movement still less; whilst internal associated movement could not be obtained at all. There was also ptosis, more pronounced on the right side.

Upon a second visit two days later, the partial external ophthalmoplegia had altered in relative amounts of muscle action, whilst the left pupil had become smaller, though the iris was still immobile to light.

Two days after this the remaining field of the right eye had become "black blind."

Dr. Hinsdale also exhibited three large sections of a brain tumor from a patient of Dr. H. C. Wood (case reported in the *University Medical Magazine*, Phila., April, 1889).

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Nervous and Mental Disease.

Original Articles.

PARALYSIS OF SERRATUS MAGNUS.

BY ROSS R. BUNTING, M. D.

T. M., aged twenty-four, white, married, presented himself, September 16, 1889, at Philadelphia Polyclinic Service of Dr. Chas. K. Mills, with the following history: June 7, 1889, at 4 A. M., while at night-work in a dye-house in Wilmington, Del., he noticed he could only raise his right arm half way to the shoulder. He had some pain in the shoulder at the time, which continued at intervals for two weeks, not only when he attempted to raise his arm, but when it was perfectly quiet. General health good. Father died at forty-five of sun-stroke; brother and sister living in good health.

Present condition: Very slight wasting of muscles of right shoulder as compared with the left. When the arm is at rest there is not much deformity. (Fig. I.)

The inferior point of left shoulder is lower than the right; the lower angle of right scapula somewhat nearer the spine than the left. With right arm elevated (Figs. II., III.), the scapula is raised up and projects behind in a wing-like manner; the inferior angle goes backward toward the spine, external angle upward and forward. He cannot thrust the scapula forward as in the left or well side. He can only raise his arm half way to the shoulder.

Farado-contractility abolished; reactions to galvanism

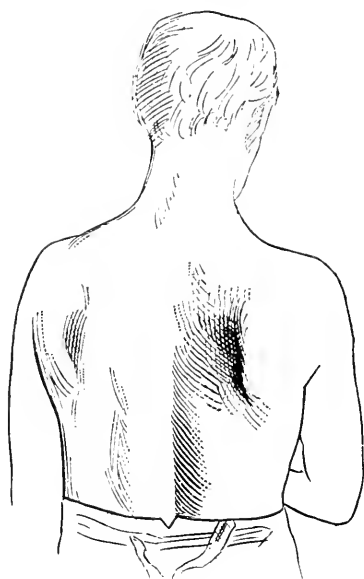


FIG. I.



FIG. II.



FIG. III.

present, but altered; modal changes; chest-wall smoother on right than on left side; insertions of serratus (sixth to ninth ribs) are not seen during forced inspiration. From the inability of the patient to raise the arm to the horizontal position, there is apparent involvement of the deltoid. "The arm is raised least by the posterior and most by the anterior fibres (of deltoid), but even the latter only elevate it to a right angle with the trunk" (Gowers). According to Allen ("Human Anatomy"): "When the entire muscle (serratus magnus) contracts, the ribs being fixed, the scapula is drawn forward (adducted) and held firmly against the thorax, thus enabling the muscles arising from the scapula to secure definite lines of traction. The most important of these is the deltoid, which cannot effectively raise the arm from the vertical to the horizontal position unless the scapula has been previously fixed by the serratus magnus."

Niemeyer says ("Text-Book of Practical Medicine," vol. ii., p. 336): "The serratus is especially required in the act of elevating the arms above a horizontal level, as it then draws the lower angle of the scapula outward and turns the glenoid cavity of the joint upward. It is by this act alone, and not by the contraction of the deltoid, that we are enabled to lift the arm above the shoulder. When the serratus is paralyzed, the inner border of the scapula, and particularly its lower angle, instead of lying against the chest, stands up like a wing, drawing up a three-cornered fold of skin before it and admitting of our reaching deeply into the subscapular fossa. The antagonistic muscles, the trapezius and levator scapulæ, have drawn the superior angle upward, and the weight of the arm and the pectoralis minor have depressed the external angle and thrown it forward."

In most of the cases reported there is a history of traumatism. Ross ("Diseases of Nervous System") reports the case of a man having fallen upon his right shoulder, in which the characteristic deformity was present. The man was able, but with some difficulty, to raise his arm above the horizontal level. Ross attributes the paralysis in this

case not to direct injury to the muscle, but to "injury and consequent neuritis of the long thoracic nerve as it passes through the scalenus medius, caused by the sudden contraction of the muscle when the patient threw out his right arm in order to protect himself while falling."

G. V. Poore ("Electricity in Medicine and Surgery") relates a case in which the patient "over-exerted himself and says he strained his right arm, and complained then and some time afterward of severe pain in right shoulder and arm. One point of great interest in this case is the time of the appearance of the paralysis, which was not till three months after the date of the mishap to which the patient attributes his troubles. The strain seems to have affected the brachial plexus, and to have caused a sub-acute attack of neuritis, as evinced by the pain and tenderness along the nerves and the congestion and sweating of the hand."

According to Erb ("Ziemssen's Cycl. of Med.," vol. xi., p. 530): "Paralysis of the serratus, both unilateral and bilateral, has not unfrequently been observed after over-exertion of the muscles of the shoulder, as in mowers, puddlers, shoe and rope-makers. These various circumstances explain why paralysis of the serratus muscle is far more frequent in men than in women, and chiefly occurs on the right side. 'Catching cold' is also a frequent cause, numerous cases having been reported in which the affection has been produced by exposure to draughts, by sleeping on damp ground or near a damp wall."

The paralysis in our patient, T. M., was evidently due to this latter cause, as it occurred while at work during a damp night. The treatment recommended was galvanism and massage and the remedies for rheumatism. The treatment of these cases is not very satisfactory, especially as regards quick recovery.

Erb says: "After having made its appearance suddenly or gradually, it may remain stationary for a variable period, often for many months, and it only very gradually terminates in recovery." This statement is verified in the treatment of the case reported. His physician, Dr. Stubbs, of Wilmington, Del., writes (November 8, 1889) that his condition is about the same as when he first presented himself at the Polyclinic.

THE KNEE-JERK AFTER SECTION OF THE SPINAL CORD.¹

By EDWARD T. REICHERT, M.D.,

Professor of Physiology, University of Pennsylvania.

SINCE Westphal and Erb introduced the knee-jerk, or the patellar-tendon-reflex, or knee phenomenon, as it is sometimes called, as a diagnostic factor it has assumed a position of questionable value, and of late years generally been looked upon with increasing distrust. Why this should be seems evident in the extensive and acute observation demanded in its study; in the uncertain character of our knowledge regarding its true nature and its full significance; and in the almost entire absence of information of the conditions, normal and abnormal, which affect it.

While the nature of the knee-jerk is not definitely known, there can be no doubt that its manifestation depends upon the functional integrity of a reflex arc connecting the quadriceps femoris with cerebro-spinal centres, and that the essential part of this is found in groups of cells constituting the proper centres in the spinal cord. Other things being equal, the extent of reaction when the tendon is struck depends: first, upon the condition of the reflex arc, which when affected, as in *tabes dorsalis*, where the sensory paths are interfered with, is diminished or lost, or as in *lateral sclerosis*, where perhaps the spinal cells are indirectly excited, is exaggerated; second, upon the degree of tension placed upon the patellar ligament, the reaction increasing with the tension up to a certain point; third, upon the strength of blow, the reaction increasing with the degree of stimulus.

Should the tendon be repeatedly struck at proper intervals by blows of like strength, the successive reactions will, as a rule, be practically of the same extent, but occasionally more or less distinctly exaggerated or dimin-

¹ Read before the American Society of Physiologists, December 28, 1889.

ished, and, at times, to a remarkable degree. These incidental or unexpected modifications in the average reaction have been the cause of some confusion, and the actual reasons for their occurrence were not apparent until the discovery of Jendr sek ("Deutsches Archiv. f. klin. med.," Bd. 33, S. 177, 1883) that if the hands were clinched immediately before the tendon was struck the knee-jerk was greater than when the individual was perfectly quiet. While this significant fact does not seem to have been fully appreciated by him, it has fortunately led to investigation in this country, which has been the starting point of observation of rare clinical importance, and which has already thrown much light upon this obscure subject.

Mitchell and Lewis ("Philadelphia Medical News," Feb., 1886), and Mitchell (*ibid.*, June, 1888) found that any movement, if at all decided, and that a vast range of sensations, if made or perceived simultaneously with the tapping of the tendon, would exaggerate the reflex; and, further, if a galvanic current were passed through the anterior part of the brain it was also increased, probably owing to "a complex effect made up of sensation, motion, emotion, and some other more immediate affections of the brain." In Lombard's studies ("American Journal of Psychology," Oct., 1887) we note most important additions to our knowledge of the variations which occur in the normal knee-jerk. He found that it suffers a diurnal variation, being highest in the morning; is increased after each meal; is diminished by muscular or mental fatigue; is increased by mental activity; is affected by the weather, being in general increased by a fall of temperature and decreased by a rise, rising and falling with the barometer, but not visibly influenced by the direction of the wind, the humidity or electrical potential of the atmosphere. In general, whatever increased or diminished the activity of the central nervous system similarly influenced the knee-jerk. Moreover, voluntary movements and sensory irritations, even when not strong enough to cause a reflex, would when synchronous with the blow upon the tendon increase the reflex. Strong emotions, even during sleep when the dreams are vivid, acted simi-

larly. Thus music, the cry of a child, a knock at the door would affect the degree of reaction.

The fact that the volitional movements, sensations, or emotions must occur simultaneously or slightly antecedent to the tapping of the tendon to reënforce the reaction, led Bowditch and Warren ("Boston Medical and Surgical Journal," May, 1888) to make some investigations to determine how the extent of the knee-jerk would be affected by varying the interval of time at which the blow on the tendon followed the volitional movement for reënforcement. As the result of 551 normal and 624 reënforced knee-jerks in the same individual, they found "that if the blow follows the signal [for a volitional movement] at an interval not greater than 0.4," the reënforcing act *increases* the extent of the knee-jerk. If the interval exceeds this amount, a *diminution* of the knee-jerk results. If, however, the interval is prolonged to 1.7, "the reënforcing act is without effect on the knee-jerk."

Thus we find that the knee-jerk is temporarily modified by many normal conditions, and that it may undergo decided momentary variations through a volitional movement, sensation, or emotion occurring at about the time of the blow upon the tendon, and that the volitional movement may reënforce or inhibit the normal degree of reaction depending upon the time of its occurrence in relation to the time of tapping the ligamentum patella.

In all the instances where incidental reënforcements or inhibitions of the knee-jerk occurred, it seems evident that the mind played an important part, either in the origination of the volitional movement or the emotion, or in the perception of the sensation. It was therefore suggested by Dr. S. Weir Mitchell that experiments be made upon the lower animals, in which the spinal cord was previously cut, to learn if similar alterations could be induced where all cerebral action would be cut off. I accordingly performed twelve experiments on dogs, which were anæsthetized and the cord cut in the lower cervical or upper dorsal region. In all cases the operation was practically bloodless, save one, where the hæmorrhage amounted to less than three

ounces; and the sections of the cord were complete, as shown by autopsies. Immediately after the section the reflexes are diminished, but in the course of a couple of hours or even less are normal, when the animals may be studied. To accomplish this, the dog is placed upon his side on the table and held gently but firmly by an assistant; the leg is suspended, the tendon struck, and the reaction recorded, as in Lombard's experiments on man. An additional record was also made, by a suitable recording-apparatus, of the time of the application of the reënforcing stimulus and that of the occurrence of the reaction. The stimulus was either mechanical, by strongly pinching the toes of the opposite foot, or electrical, where the electrodes were thrust into the skin of the opposite leg or into the upper part of the peripheral segment of the spinal cord, and an induction-current used of sufficient strength to cause pain when applied to the hands. More or less trouble was experienced in keeping the animals quiet, to prevent a disarrangement of such a complex piece of apparatus and avoid inaccurate records, but with much care it was found in all cases, without exception, that there was never any positive evidence that the knee-jerk was affected in any way by these conjoint excitations, no matter what their true relations were in conjunction with the blow upon the tendon.

These uniform results therefore indicate that the reënforcement or inhibition of the knee-jerk are dependent upon some peculiar influence exerted by the cerebral centres.

A CASE OF ACUTE MELANCHOLIA, DURING
THE PROGRESS OF WHICH THERE AP-
PEARED ARGYLE-ROBERTSON PUPIL,
WITH ABOLISHED PATELLAR RE-
FLEX ON ONE SIDE AND MUCH
DIMINISHED ON THE OTHER.¹

By H. A. TOMLINSON, M.D.,

Assistant Physician to the Friends Asylum for the Insane, Philadelphia.

MR. J. P. D. was admitted to the Friends Asylum, as a patient, July 3, 1889, with the following history: Is 49 years old, married, and has two children. Born and lives in New Jersey, and is a bank-teller by occupation.

In his younger days he worked at the trade of carriage painter and trimmer. He gave his trade up after a number of years on account of his health, and went to farming. After six years he gave this up also, because it was too hard for him, and took a position as teller in a bank. He has always been a very industrious and morbidly conscientious man, inclined always to look on the dark side. For the last ten years he has been working beyond his strength, and has suffered very much during the most of that time from indigestion. He has had occasional attacks of depression, more or less severe, and during these attacks of depression has suffered from insomnia and anorexia.

For a long time Mr. D. had been keeping the accounts of two other organizations besides his regular clerical work at the bank, and about three months ago began to work at night also. He began now to suffer from insomnia and constipation, from which he had always suffered; became worse, so that he was obliged to take medicine to relieve it. He did not care for food, and what he ate only added to his discomfort. From being a quiet, considerate and

¹ Read before the Philadelphia Neurological Society, December 23, 1889.

self-contained man, he became changed to an irritable, restless and peevish one. He began to manifest unusual and unreasonable annoyance about small business trifles, and became intensely depressed and despondent.

About this time he discovered a small error in his accounts, and began to dwell upon it, until he came to believe that he had involved himself, that his sureties would be held responsible for his error, that it would take all he possessed to reimburse them, he would be left penniless, and would not be able to provide for his family.

These delusions have persisted and become more perfectly defined, until now no amount of persuasion or argument has any influence in dislodging them.

He was under active treatment for two months previous to his being brought to the institution, but although his physical condition improved somewhat, his mental condition grew worse, until finally his family brought him to the institution for treatment.

At the time of his admission he was found to be taciturn and disinclined to answer questions. There was also marked failure of memory, with confusion on attempting to express himself on general subjects; but he did not hesitate to talk about his delusions, dwelling most particularly on those relating to his property. His physical condition was very bad. He was pale and much emaciated, his countenance expressive of anxious distress.

He is a small slight man, with brown hair and eyes and high narrow and prominent forehead. He is narrow-chested, stooped, and with the left shoulder higher than the right. Tongue pale and flabby, with slight coating in the centre. Pulse 120; temperature $98\frac{1}{2}^{\circ}$; heart-sounds normal, but weak, no dilatation; lungs normal; respiration 24. Examination of the urine gives a negative result, the only abnormal constituent being a slight excess of urates. His bowels were moved the morning of his admission.

During the first two days of his residence in the institution his condition remained unchanged, excepting that he grew weaker; at the end of that time he became restless

and excited, and was most of the time in a profuse perspiration.

On the evening of the third day after his admission he asked his attendant for a knife to kill himself with. He was put to bed and placed upon liquid diet. He was better the next morning, and his pulse came down to 108. His pulse was peculiar, in that it had a hard feeling, as if there was a considerable degree of arterio-sclerosis; but a careful study of the pulse showed this condition of apparent hardening to be due probably to vasomotor spasm. Mr. D's physician had stated that this condition of the pulse had existed for a long time previous to his coming to the institution. He began at this time to grow apprehensive about his family, feeling that his conduct had, or would, destroy them. He became more excited, and developed auditory and visual hallucinations, seeing and hearing people, with whom he had been familiar, in his room.

During the next few days his condition remained practically the same, excepting that his pulse fell to 98. His bowels began to move every morning, and he slept a little better. About this time it was noted that Mr. D. had three periods of marked depression, very nearly uniform, during the day, and lasting about two hours—occurring after he waked in the morning, in the middle of the day, and in the evening. The depression was most profound on waking.

At this time a hot bath, followed by friction, and given at bed time, was added to the treatment. He was also given a wineglass of malt every three hours. During the next week his general condition remained comparatively unchanged. He has developed a new delusion, to the effect that when he came here he assumed the responsibility for the expenses of the institution and its management; and one to the effect that the other inmates and the attendants did not get enough to eat. His original delusions have been dominated by these last two new ones, excepting the delusion of poverty, which is merged in that of his responsibility. During the next week there was no change in Mr. D.'s mental condition nor the character of his delusions. In the early part of the week he had a

crapulous diarrhœa, caused by undigested food. He had been eating at table, and his mind was so occupied with his delusions that he did not pay enough attention to his meals to chew his food properly. He was placed upon a special diet and the diarrhœa ceased in a day or two. He had slept fairly well and his pulse has ranged from 84 to 96. Two different times during the week Mr. D. was rational for a couple of hours. His delusions did not change as to subject and were the rest of the time as active as ever. Physically, he had been gaining steadily, but very slowly.

At this time attention was called to the fact that, even in twilight, his pupils were minutely contracted. Examination showed that there was no reaction to light, but accommodation was not affected. There was no tremor of the tongue. The patellar reflex was entirely abolished on the right side and much diminished on the left. There was also quantitative electrical change. During the next two weeks his condition remained about the same, except that his physical condition slowly improved, while at the same time he became restless and generally distressed. His delusions, though unchanged in subject or character, were less active. His urine was examined again and found normal. The condition of the pupils and reflexes remained the same. His pulse during this time kept at about 96 and was of the same character as at first. During the next few days the restlessness and excitement increased, so that unless he was compelled to sit still he was constantly on the move. He would not eat unless urged, because he thought that by so doing he was depriving the other patients and the attendants of food. He was very much disturbed also because he thought that people in the building were being starved and injured or killed, and he tried to keep himself awake at night, because he felt that he ought not to sleep when such terrible things were going on. He gradually lapsed into a condition of typical melancholic distress, with haggard, anxious countenance, hurried respiration and intense restlessness—keeping up his tramp, tramp, backward and forward, in his room from morning until night, and, if not watched, would get up in the night

and tramp also. During this time he lost all he had gained in weight, and became, as a matter of course, much emaciated and exhausted. The eye ground was examined at this time, but with negative result, only a little narrowing of the arteries being found, which corresponded to the condition of the radial arteries. Refractive error was also noted. There was noted a considerable degree of ankle clonus on the right side at this time also. During the existence of this intense excitement there was no interference with digestion and his bowels were moved regularly. This condition lasted for about ten days, after which it began to subside, and with its subsidence came a marked change in his general condition. His pulse from being, during this distressed period, 110 to 120, small, hard and feeble, fell to 72, and became full and soft. The reflex iridoplegia disappeared, as did also the ankle clonus, and the patellar reflex began to improve. His physical condition improved also.

At this time his delusions began to change. From being troubled about the people in the house, he began to be troubled about his family, and talked about them a great deal—accusing himself of great wickedness and of failure in his duty toward his family, believing that his conduct had ruined them. Another delusion which developed at this time and continued to the last, was to the effect that he had assumed the responsibility for the management of the institution, and that by doing so he had wilfully made his sureties responsible for his conduct, and that he had no means to compensate them. This was a modification of one of his original delusions to the same effect, but which had been for the time being lost sight of. It will be seen that all of his delusions hinged upon and were built up from his original delusion of poverty and responsibility, they being changed by the influence of his new surroundings into the different forms in which they were manifested during his residence in the institution. The only thoroughly systematized delusion was the one concerning his bondsmen's responsibility for the management and expenses of the institution, and this persisted until within a week of his being discharged restored.

After this he began gradually but steadily to improve, both physically and mentally, and although the excitement and distress continued in a modified degree, he began to be willing to discuss the possibility of his delusions being without foundation. He talked more about his family affairs also. He had, however, still to be urged to eat, especially meat, which he thought was the flesh of some one who had been killed in the house. The rest of his meal he would eat voluntarily. Oysters and fish he would also eat with but little urging. About this time he began to believe that his wife was shut up somewhere in the institution. He continued, however, to write to her at home, not recognizing any incongruity in his conduct. He kept in this condition for about ten days without much change except steady physical improvement. At the end of this time he suddenly began to brighten mentally and for the first time, admitted without reserve the groundlessness of his delusions. He did not, however, give them up entirely, but was willing to talk about them as being delusions. He now became very anxious to go home, but was full of fear that his townspeople knew of his ill-doing and would look upon him as a criminal. He also began to be interested in his surroundings and spent most of his time out of doors. In another week he had given up his delusions entirely and became quite cheerful. He now began to take his meals at the officers' table, and was given the freedom of the grounds. He availed himself of this freedom, and spent his time in roaming about the neighborhood. During the next week his mental illness disappeared entirely, and being practically well, he was discharged. Before leaving the institution he was sent to an oculist, who found that he had a mixed astigmatism with hypermetropia, for which condition he was furnished correcting glasses.

There was nothing especial in the treatment of this case so far as drugs were concerned to refer to. During the first two weeks he had no drugs at all. At the end of that time he was put upon a pill of arseniate of strychnia, digitalin, zinc phosphide, quinine and bromide of iron, which he took continuously during his residence in the institution. He

also had hot baths, followed by friction. These baths were very efficient in securing sleep at night, and combined with massage and Swedish movements, stimulated the circulation, kept the skin active, and aided materially in improving nutrition. He also had general faradization. This routine of treatment, combined with a carefully selected diet, and milk and malt between meals, was kept up from the second week, until the advent of the melancholic distress. At the end of the sixth week of his residence he became so much exhausted at this time that the baths and friction, the faradism and Swedish movements were discontinued. The massage was kept up at bed-time to aid in securing sleep. No hypnotic medicine was given him at any time.

During the period of extreme distress, about ten days, he was given caffein and codeia with apparently good effect. It was while taking these drugs that his pulse fell from 110 to 72 and changed in character. As soon as he began to get quiet they were discontinued, and he had no medicine afterward except the pill before mentioned.

There are several points of special interest in this case to which I would like to call attention. In the first place, the patient's condition on coming to the institution, the extreme exhaustion and emaciation, the intense depression, with a history of long-continued anorexia and impaired digestive power—all these conditions combined, making it probable that even if the patient did not speedily die of inanition, his residence in the institution would be a prolonged one. Next, the entire absence of the ego in any of his delusions, while at the same time his self-accusations approximated very nearly to that delusion which always suggests a bad prognosis the commission of the "unpardonable sin." Lastly, the appearance, at the end of the fourth week, of the "Argyle-Robertson" pupil and abolished patellar reflex, and their persistence for a period of more than six weeks. This condition of the pupil would, of course, suggest paresis, but the absence of tremor of the tongue and the abolition rather than the exaggeration of the patellar reflex, pointed toward locomotor-ataxia; but there were no pains, stag-

gering gait, or inability to walk with the eyes closed. The conclusion reached at the time was that these symptoms indicated a condition of general ataxia due to exhaustion and malnutrition, and not a specific condition. Another singular symptom was the character of the pulse, which suggested immediately atheroma and kidney change, both of which, however, were absent. This condition of the pulse could be accounted for on the same hypothesis as the pupillary condition, both being due to sympathetic irritation. After the period of distress had passed, and he became more conscious of his surroundings and the possibility of his continued illness, there was redeveloped the suicidal impulse which had existed in the beginning. He tried very hard to mask these impulses when they came, and there were several occasions when if an opportunity had offered he would have made way with himself. He referred to this after his recovery, and said that he had kept it to himself purposely, so that if he found that he was not going to get any better, no suspicion having been excited, he would have a better opportunity to carry out his intention of self-destruction. However, his intention *was* suspected, and he was carefully watched always.

TREPHINING FOR EXTRA-DURAL HÆMORRHAGE.¹

By JOHN B. DEEVER, M.D.,

Demonstrator of Anatomy and Lecturer upon Surgical Anatomy in the University of Pennsylvania; Attending Surgeon to the German, Philadelphia, St. Mary's, and St. Agnes' Hospitals, Philadelphia, Pa.

APPARENTLY much has been done in the past two years in brain surgery, but by far the greatest number of operations and reported successes have been for focal or Jacksonian epilepsy. In this class of cases the immediate results have been good, but can we say this of the remote? No; and therefore in my judgment beyond giving temporary relief, or beyond holding the trouble in abeyance for a time, these operations have accomplished most in demonstrating the amount of manipulation the brain is capable of permitting, providing that this is done by skilled hands and that every possible antiseptic precaution be taken. The lateral ventricles have been tapped, but this offers no more than temporary relief. It does not by any means remove the factor, which is slowly, but surely, killing the patient. This is true to a great extent in the cases of tumors, as we know the most common forms of brain tumors are tubercular, sarcomatous, and syphilitic, and therefore in the event of their successful removal the patient is not restored to a normal condition, but is left crippled. More favorable results have been obtained in tumors springing from the dura mater or agglutinated membranes, which are not strictly tumors of the brain substance, but invade it by pressure, excavating for themselves a bed or nest. Contrast with the above, trephining for intra-cranial hæmorrhage, particularly of the extra-dural variety. Here the operation, if done early, is in every sense a life-saving agent, practical as well as permanent in its results in the majority of cases.

We have four varieties of intra-cranial hæmorrhage :

1. Where the blood finds its way between the

¹ Read before the Philadelphia Neurological Society, January 27, 1890.

inner wall of the cranium and the dura mater. 2. Where it finds its way into the sub-dural space. 3. Where it finds its way into the sub-arachnoid space and into the meshes of the pia mater. 4. Where it escapes into the substance of the brain or into its ventricles.

Erichsen gives the third variety as the most common form of intra-cranial hæmorrhage, while Prescott Hewitt gives the second variety as the most common. In the cases which I report to-night, and in which I trephined, all were of the extra-dural variety. When blood is poured out between the dura mater and the bone in cases of fracture, the vessel or vessels that are, as a rule, torn, are the middle meningeal artery or its branches. Mr. Jacobson has shown that the branches of this artery are more frequently ruptured than the trunk. The vessel is very frequently torn at the point where it crosses the anterior inferior angle of the parietal bone. The reasons for this are: 1. The bone at the site of the groove for the artery is very thin. 2. The vessel is so frequently buried in the bone that fracture without laceration of the vessel is hardly possible. 3. This part of the skull is peculiarly liable to be broken.

It has been shown that the artery may be ruptured by force sufficient to occasion detachment of the dura mater, but not great enough to fracture the skull. Next to the middle meningeal the most frequent source of extra-dural hæmorrhage is the lateral sinus. In referring to the attachment of the dura mater to the cranium, we find that it is intimately adherent to the bone forming the interior of the wall of the skull, therefore this variety of hæmorrhage in this situation is barely possible. Over the vault of the cranium its attachments are comparatively loose, except along the lines of the sutures. Sir C. Bell has pointed out that the dura mater of the vault may be separated from the bone by the vibration produced by a blow. Strike the skull of the subject with a heavy mallet; on dissecting, you find the dura mater to be shaken from the skull at the point struck. Repeat the experiment on another subject, and inject the head minutely with size injection, and you will find a clot of injection lying between the skull and the

dura mater at the part struck, and having an exact resemblance to the coagulum found after violent blows on the head. Tillaux has demonstrated, that the adhesions between the dura mater and the bone are particularly weak in the temporal fossa, the most usual site of meningeal hæmorrhage.

In the diagnosis of extra-dural hæmorrhage the following points are to be observed :

Mental Condition.—May be normal, or there may be cerebral irritation. Unconsciousness complete or incomplete, or coma.

Condition of Pupils.—May both respond to light normally. May both be dilated and show no response ; or one may be widely dilated and the other normal. When the dilated pupil corresponds to the injured side, it is caused, as pointed out by Hutchinson, by the pressure of a large clot, extending deeply down into the base of the skull, on the cavernous sinus, leading to fullness of the vessels, with protrusion of the eyeball and dilatation of the pupil. It is also accounted for by compression of the oculo-motor nerve by the clot.

Respiration.—May have stertorous breathing, Cheyne-Stokes respiration, or cyanosis.

Pulse.—Little changed, or rapid and feeble, depending largely on the severity of the concussion ; or slow and full, depending upon the severity of the compression.

Limbs.—May present any of the following conditions : Hemiplegia, well or but little marked. On the same side as the one injured, indicating hæmorrhage on the opposite side ; monoplegia, paralysis, twitching, convulsions, rigidity (spastic).

Scalp.—Presence of contusion, or boggiess due to the injury ; the latter also due to leakage from within the cranium through a fissured fracture.

The stages presented by a typical case of extra-dural hæmorrhage are three :

I. Complete or partial unconsciousness, the result of the concussion or shock, caused by the fall or blow, as the case may be.

II. Consciousness or lucidity. This stage may vary in length from a few minutes to several hours. "Is present in about one-half of the cases," says Jacobson.

A very large hæmorrhage may produce compression at once, as I have observed, verifying my observation by post-mortem examination. Compression may also come on immediately, caused by coexisting depression of bone, injury to the brain, and alcoholism.

III. Compression.

CASE I.—A. P., aged eighteen years, was admitted to the German Hospital, December 17, 1886, with pistol-shot wound of the left temporal region, the history of the case being as follows: While A. P. was playing with several companions, one of them with a single barrelled 22-calibre pistol, attempted to shoot over his head, when the hand in which the pistol was held being about twelve inches away from the side of the head, was drawn down by another companion, and the pistol accidentally discharged.

Upon probing for the bullet, the external table of the skull, at the point corresponding to the wound in the scalp, was found denuded of its periosteal covering and slightly roughened. The patient at the time of admission presented no head symptoms, was perfectly conscious, and suffered no more pain than usually accompanies wounds. The wound was dressed antiseptically, and the patient put to bed. Upon making my visit through the wards on the following morning, December 18th, I found that he was aphasic. I advised operative interference, to which he gave his consent.

He was taken into the clinic, and in the presence of the class I trephined. The outer wall of the cranium, at a point measuring two inches posterior to the external angular process of the frontal bone and two inches above the zygomatic arch, was exposed by dissecting up a horseshoe-shaped flap, from the scalp with its convexity directed downwards and backwards. This was the point of the skull with which the ball came in contact, and being immediately over the base of the second and third frontal convolutions (the portion of the hemisphere in relation to the ascending limb of the fissure of Sylvius, and the lower extremities of the fissure of Rolando, Broca's convolution, or the region of motor speech). The roughening which was detected at this point was caused by a small portion of the bullet imbedded in the outer table of the skull. The remaining part of the bullet was found one inch behind and below this point. It

lay beneath the temporal muscle and was somewhat flattened. The trephine included the roughened portion of the skull. Upon the removal of the button of bone, which demonstrated that the internal table was fractured, but with no depression of the fragments, was seen a clot, evidently the result of a rupture of the posterior of the two branches of the anterior division of the middle meningeal artery; this was removed, and as there was no further bleeding, the flap was adjusted, and the wound drained with catgut. The patient made a complete and uninterrupted recovery. The aphasic symptoms disappeared shortly after the operation.

CASE II.—J. E., aged thirty-four years, white, was admitted to the German Hospital, September 23, 1889, at 5 P. M., when he stated that he had fallen down a hatchway, a distance of twenty feet.

His condition on admission was as follows: Perfectly conscious, but a little dull mentally; he was able to narrate the occurrence of the accident very intelligently. Respiration 14, temperature 97; slight lacerated scalp wound of the right side over the parietal eminence; also contused and lacerated wound of the left elbow, opening up the capsule of the joint. He had perfect control of the bladder, being able to pass his urine shortly after he was admitted to the hospital. The wounds were dressed and the patient put to bed.

6 P. M.—Dr. Michel, the resident surgeon, was sent for to see the patient as he was quite restless, showing some evidence of cerebral irritation.

7.30 P. M.—Respiration somewhat noisy and labored, and this condition was shortly followed by a slight twitching of the right leg, arm and face, which continued with very little change until 8 P. M., when it was followed by convulsions, commencing in the right leg and becoming rapidly general.

I was sent for, seeing the patient at 9 P. M. I immediately explored the scalp wound, but could find no evidence of fracture. I removed a button of bone, two inches in diameter, from the left side of the skull, the centre of the button corresponding to that of the scalp wound, which I believed to be the point of the greatest intensity of shock from the fall, and where I therefore expected to find the maximum amount of separation of the dura mater, and consequently the largest amount of clotted blood, in the event that I was correct in my diagnosis of extensive but circumscribed extra-dural hæmorrhage. Upon the removal of the button of bone, clotted blood immediately

commenced to escape through the trephine opening. I removed all of the clot, leaving a space between the skull and the dura mater, which held four ounces and one drachm of a 1-2000 bichloride solution. Rubber drainage tube, of medium size, was introduced into the bony opening, and brought out at the lower angle of the scalp wound. The wound was adjusted with silver wire sutures, and the head dressed. The patient lived but a short time after the operation, not having reacted.

Autopsy made the following day, December 26, 11 A. M. The brain and cranial cavity alone examined. Upon the removal of the calvarium the surface of the dura mater was free of blood and otherwise normal, except at the point of separation already referred to. Brain removed. Dura mater lining sides and base of skull removed, when was seen a fissured fracture starting in left parietal bone immediately below opening made at operation, and extending into the floor of the middle cerebral fossa, involving only the outer half of same. Examination of brain showed it to be perfectly normal except at three points on the basilar aspect of the temporo-sphenoidal lobe; these were evidently seats of contusion, involving only the gray matter, as shown by making sections of the brain. No further evidence of cranial hæmorrhage or other lesions.

CASE III.—Harvey Kitchen, twenty-three years old, white, was admitted to the German Hospital, December 19, 1889, 12.30 P. M., having met with an accident. He had fallen a distance of fifteen feet through an elevator, landing on the top of his head.

His condition upon admission, as described by the resident surgeon, Dr. Gerlach, was as follows: Complete unconsciousness, pupils responding to light, conjunctival reflexes active, respiration 24, pulse 100, temperature 99°. The scalp was much swollen and boggy over the whole of the left side and vertex, with lacerated wound of the right ear at the junction of the concha with scalp. The urine was drawn; quantity small, normal in reaction and color; chemical tests for albumen and sugar negative. The patient remained in the above condition twenty minutes, when he regained partial consciousness for about five minutes; he was then seized with a general epileptiform convulsion, lasting one minute. The convulsion, as described by the Sister, was thought to have commenced in the right leg, but it became general so soon that she could not be certain. After the convulsion the patient could not be aroused fully, but showed some evidence of consciousness when spoken to. His breathing was embarrassed, the pulse full and

strong. After an interval of ten minutes he was seized with another convulsion, lasting about double the length of the first, and described by a second Sister present as corresponding with that of the first seizure; but she too was in doubt about the spasm commencing in the right leg, as it became general so soon. Dr. Gerlach was sent for, but arrived too late to witness the convulsive seizures. The patient was now intensely cyanosed, respirations slow, deep, and stertorous, pulse slow and full. Upon my arrival at the hospital, but a moment or two later, very fortunately, I found his condition as above noted. Examination of the right side demonstrated the limbs to be slightly spastic, the left parietic.

As there was but little doubt in my mind that the condition was one of intra-cranial hæmorrhage, and most probably of the extra-dural variety, there having been an interval of consciousness between the reaction from the concussion or shock and the development of convulsions, I immediately prepared to trephine over the anterior branch of the middle meningeal artery. The scalp was quickly prepared for a strictly antiseptic operation, and ether administered. A large horse-shoe flap, including all the layers of the scalp, with its convexity directed downward and backward, was turned up on the left side by carrying an incision from directly behind the external angular process of the frontal bone to above and behind the parietal eminence; then was seen a fissured fracture following the coronal suture. To trace it throughout its entire length, two further incisions were made, one following the line of the coronal suture, the other from the termination of the latter, a short distance over the right parietal bone, when these flaps were reflected forward and backward, almost the entire vertex was laid bare.

In the subaponeurotic space and infiltrating the areolar tissue of the scalp was an extensive extravasation of blood. The fracture extended from the beginning of the coronal suture to about one and a half inches beyond the sagittal suture; from this point a fissure extended obliquely downward and backward into the right parietal bone, also one obliquely forward, and to the left, involving both the right and left frontal bones, stopping short of the supra-orbital margin. There was some bleeding from beneath the bone, through the line of separation of the coronal suture, most marked on the left side. With a medium-sized trephine, an inch and a half in diameter, a button of bone was removed at a point one inch and a half behind the external angular process of the frontal bone; and one and three-quarter

inches above the zygoma, when the anterior branch of the middle meningeal artery was exposed and its ends ligated with catgut. The bleeding not being entirely arrested, with a pair of Hopkins' modification of the rongeur forceps the opening was enlarged in the direction of the groove in the squamous portion of the temporal bone, through which passes the posterior branch of the above vessel, when it was plainly to be seen that the source of the remaining hæmorrhage was most probably this branch near its point of origin; the opening was therefore enlarged in the direction of the spinous foramen of the sphenoid bone, and after cutting away bone, to the extent of about one-half inch, the vessel was seen to be torn and bleeding freely. Both ends were tied with catgut, after which the hæmorrhage ceased. The cavity occasioned by the separation of the dura mater was washed out with a 1-2000 bichloride solution.

I removed a second button of bone, three-quarters of an inch in diameter, from a point about three-quarters of an inch to the left of the sagittal suture, and about one inch posterior to the separated coronal suture, this corresponding to the upper and posterior margin of the cavity caused by the separation of the dura mater. My object in doing this was to enable me to pass catgut-drainage through this opening and out of the one first made, this securing a perfect outlet to any liquid that would otherwise have a tendency to collect in the cavity. Through this last opening I incised the dura mater and exposed the sub-dural space, to determine whether or not there was either the second or third variety of intra-cranial hæmorrhage associated with the extra-dural which I had already checked. This proved negative. The incision of the dura mater was closed with catgut suture. The end of a rubber drainage-tube, one-quarter of an inch in diameter, was placed in the lower and anterior of the two openings made in the wall of the skull, and brought out at the lower angle of the scalp-wound, where it was fixed by a silver wire suture, loosely twisted. A second rubber drainage-tube, the same size as the above, was placed beneath the scalp-flaps, above the two trephine-openings, and brought out in front and behind, where it was fixed by a silver wire suture. The flaps were now completely adjusted and held in apposition by interrupted silver wire sutures. Through the tubes a 1-2000 solution of bichloride of mercury was passed, and the wound dressed antiseptically. A hypodermic injection of one-eighth of a grain of morphia was given to the patient, when he was removed from the operating-table. Dry heat was applied to the body and an ice-bag to the head. He reacted well,

and slept for four hours after the operation, when he awoke and was fairly rational. Being now able to swallow, he was given milk diet and ordered one-quarter of a grain of calomel, with two grains of Dover's powder every two hours. At 8 P. M., six and one-half hours after the operation, he was perfectly quiet and entirely rational. Pulse 96, respiration 28, and temperature 100°; pupillary reaction normal. He was able to execute the ordinary movements with his extremities; said he felt pretty comfortable, although he had some pain in his head; during the night was somewhat restless.

December 20th.—Condition unchanged from that of previous evening.

At 3.30 A. M., December 21st, he had a slight convulsion, beginning in right upper extremity, with flexion of hand upon wrist; then forearm on arm; when it immediately became general; this lasted about one-half minute. At 5.30 A. M. had second convulsion similar to the above and lasting about the same length of time. Between and after these convulsions he slept.

8 A. M.—Condition of patient good, with the exception of pulse, which was full and bounding. In addition to the calomel and Dover's powder, tincture of verat. virid., two drops, and bromide of sodium, twenty grains, was ordered every two hours.

December 23d.—Dressings changed; no pus; drainage-tubes clear; tincture of verat. virid. and bromide of sodium stopped.

December 26th.—Calomel and Dover's powder stopped since slight mercurial stomatitis had developed, for which a gargle of chlorate of potassium and listerine were given. From this on the recovery was uninterrupted.

[This patient was exhibited in perfect health to the Society.]

In answer to the question, what led me to trephine in these cases, I will simply make a summary of the main symptoms of each:

CASE I.—(1) The injury to the outer table of the skull, as demonstrated by the probe, near the point corresponding to the location of the anterior branch of the middle meningeal artery; (2) aphasia coming on a few hours after the accident.

CASE II.—(1) Circumscribed contusion of the scalp; (2) period of consciousness following the accident; (3) evi-

dence of cerebral irritation with twitchings of the side of the face and of the extremities opposite to the side injured; (4) convulsions commencing on the side opposite to that injured; (5) coma; (6) paralysis.

CASE III.—(1) Unconsciousness; (2) pupils responding to light; (3) conjunctival reflexes active; (4) period of partial consciousness; (5) convulsions.

In closing this paper, I would not have you understand, from what was said in the beginning, that I am opposed to trephining for focal epilepsy, brain tumors, etc. I am not; but I do not consider the results in these cases as brilliant as in cases of extra-dural hæmorrhage.

As the subject of head injuries, both in their immediate and remote effects, is of interest to the neurologist as well as to the surgeon, it may not be out of place to tabulate my rules for trephining in this class of cases.

In this paper I have given the conditions for which I trephine when I suspect extra-dural hæmorrhage.

I trephine also in simple depressed fracture of skull, with or without symptoms of compression.

In compound depressed fractures, with or without symptoms of compression.

In punctured or incised fractures.

In fractures of the orbital plate of the frontal bone.

In punctures of the cribriform plate of the ethmoid bone, applying trephine to roof of orbit at the inner angle, thus opening up the most dependent part of the anterior cerebral fossa, and thereby favoring drainage, which is a chief indication in this class of injuries.

For foreign bodies in the brain.

For impacted fractures.

In gunshot injuries of skull.

In contusion of the scalp, simulating depressed fracture; here exposing the cranium at the seat of contusion, when, if depressed fracture is found, I trephine; and also, if a fissured fracture, if there is any hæmorrhage through the line or lines of fracture.

TWO CASES OF PARALYSIS OCCURRING DURING THE PUERPERAL STATE.¹

By M. IMOGENE BASSETTE, M.D.,

Instructor in Electro-Therapeutics and Clinical Assistant in the Philadelphia Polyclinic
Clinician to the Woman's Hospital, Philadelphia.

AS cases of paralysis seemingly related to the puerperal state are comparatively rare, and yet of interest from the point of view of causation and prognosis, it might be worth while to record with a few remarks two cases which I have had the opportunity of observing in the Polyclinic service for nervous diseases.

CASE I.—Mrs. McC., aged twenty-two, about four months before coming to the clinic had given birth to her first child. The labor was easy and short. The uterus was entirely freed from clots, and convalescence was normal. Milk appeared in thirty-six hours and the lochia were sufficient, gradually ceasing after the thirteenth day. She had no fever. Three weeks after labor the patient seemed to be doing well; she ate her breakfast as usual. An hour later, however, she was completely paralyzed both as to motion and sensation on the right side and also was aphasic, but she was not unconscious at any time. Sensation was diminished on the left side, particularly in the thorax and arm, but returned to normal on this side in fourteen days; it gradually improved to some extent on the right side, commencing in the lower extremities. Her speech had also slightly improved. She had no eye symptoms. Slight swelling of the hands and feet lasted about two weeks.

When she first came for treatment she had paresis of the right upper extremity, most decided in the forearm and hand, the grasping power of the hand being particularly affected; she had also marked anæsthesia over a portion of the right half of her body, the loss of sensation being to pain, touch and temperature. The anæsthesia was present in the right arm, the right half of the trunk, back and front, to a line about two inches above the anterior superior spinous process of the ilium. Another anæsthetic area, oblong in

¹ Read before the Philadelphia Neurological Society, Dec. 23, 1889.

shape, was situated in the central portion of the lower half of the front of the thigh. Another area was on the posterior aspect of the leg from the knee to just above the ankle. Other portions of the body and limbs were sensitive to impressions. From the history obtained from the patient's husband, and imperfectly from herself, she had apparently been completely hemianæsthetic at first, and sensation had gradually returned, except in the parts just described.

She presented a decided form of motor aphasia. She could understand all that was said to her, which she indicated by the expression of her face and gestures. When given something to read, she appeared to understand it, but she used only two or three expressions for everything she wished to say. She could name some things spontaneously, but not nouns and verbs; and she could utter many words which were repeated to her. She evidently received auditory and visual impressions, and turned them in her mind into names and ideas, but she could only speak a few simple words.

She remained under observation and treatment for ten months, during which time she made very great improvement in her paralysis, anæsthesia and aphasia. For the anæsthesia faradism with the dry brush was used; and a faradic current with moistened electrodes was used for the motor paralysis. For the aphasia a system of persistent training was undertaken, and met with considerable success. Internally, she was given potassium iodide and tonics. When she discontinued treatment, the anæsthesia and paralysis had almost completely disappeared, and the aphasic condition was greatly improved.

CASE II.—Mrs. M. E. P., aged thirty-eight, white, married, was perfectly well until her last labor, which occurred July 3, 1888. Her labor was normal, and she was well for eight days. On the eighth day the nurse was absent, and the patient sat up most of the day. When the nurse returned she found the patient standing up, reeling and trying to get into bed, which she managed to do, and then became insensible. On coming out of this condition several hours later, she was unable to speak or to put out her tongue, but was not otherwise paralyzed. She remained in bed, and a week later was taken with a severe chill, but did not lose consciousness. It was found then that she was paralyzed in the right arm and leg. In three or four weeks she began to use the paralyzed side a little, and gradually became well enough to walk about with some assistance.

When she came under observation, her face was drawn to the right side; she could wrinkle her forehead and close the right eye; and the tongue and uvula turned a little to the right. The right arm was a little smaller than the left from the shoulder to the hand. She could not raise her arm, which was completely paralyzed and the fingers contracted and flexed at the second phalangeal articulation. The thumb was flexed and drawn in over the palm of the hand. The right leg was completely paralyzed. Knee-jerk was exaggerated, and ankle clonus present in the right side. On the left side the knee-jerk was slight and there was no ankle clonus. Tactile sense was blunted on the right side. No cardiac murmur was present.

This patient remained under observation only a few days. The first case was treated for many months. I have thought it worth while, however, to notice the second case, as it corresponds in many points with the first, and both probably belong to the same category. In both the attacks came on comparatively soon after labor, and were probably connected with the puerperal state. Heart disease was absent in both cases; in both the involvement of speech, face, arm and leg, and the presence of contractility to electricity indicated a cerebral origin for the paralysis.

The literature of the subject of paralysis following labor does not appear to be extensive. The most recent paper is that of Dr. Lloyd in Hirst's *American System of Obstetrics*. This writer refers to the papers of Hervieux, Churchill, Imbert-Gourbeyre, Poupon and others. I have also found a few references to special cases. Lloyd objects to the term puerperal paralysis, if anything distinct or special is meant by that expression. The term, however, is a proper one for cases occurring at or near the time of labor, if it can be shown that the palsy has any connection with the process of labor or the phenomena of the puerperal state. The term is of course used only as we speak of other forms of paralysis with reference to their etiology. It is interesting to discuss the probable manner in which the monoplegia and hemiplegia which follow labor originate. Formerly uræmia was regarded as the most frequent cause.

Dr. Dercum and others have reported cases of hemiplegia occurring in Bright's disease apparently without any vascular or other lesion of the brain; in other words, due to some unilateral action of the poison on the nervous system. The truth probably is that some of the cases of so-called puerperal monoplegia and hemiplegia are uræmic in origin, but only in cases with chronic nephritis, and equally chronic disease of the blood-vessels. In both of

the cases reported no evidences of disease of the kidneys was present when the patients came under observation, but this was a considerable time after the occurrence of the paralysis. Nothing in the history of these cases indicated the probability of uræmia. In both the labor was normal, and in both the patients were doing well up to the time the paralysis occurred. The first patient was an unusually healthy looking woman; the second was pale, anæmic, and not strong looking. Uræmia can probably be excluded as the cause in both the cases.

In healthy women paralysis may occasionally occur owing to the great strain which is put on the blood-vessels during labor. Still, this is very rare, the system seeming to adapt itself to what it has to bear. In women with diseased blood-vessels associated with nephritis, such a result may of course readily occur. There would seem to be no particular reason why hæmorrhage should occur during the puerperal state unless as the result of increased blood pressure.

Hysterical hemiplegia may occur after labor as under almost any other circumstances. These two cases, and especially the first, seem to present some features of hysterical paralysis. The patient was hemianæsthetic as well as paralyzed. Against the view that the case was of hysterical nature are the facts that she had an aphasia of the kind usually associated with destructive brain disease; the persistency and peculiarity of the distribution of the anæsthesia; the absence of affections of the special senses, and also the absence of the moral and psychical symptoms which are usually present in hysteria. The fact that the patient improved but did not recover is in favor of the organic affection. Hysterical hemiplegia and hemianæsthesia are more likely to occur on the left side of the body, but of course a point of this kind is not of great value. Hysterical aphasia occasionally occurs, but it is rare and usually recovered from soon. The fact of the occurrence of organic aphasia only indicates that embolic paralysis which is probably that which most usually occurs after labor is most likely to be the result of blockage of arteries on the left side of the brain.

MORPHINE INJECTIONS FOLLOWED BY EMPHYSEMATOUS GANGRENE (MALIGNANT ŒDEMA).

By L. BREMER, M. D.,
ST. LOUIS, MO.

PRELIMINARY REMARKS.

ONE of the varieties of septicæmia well known to, and dreaded by, the surgeons of former generations, but now almost banished from the surgical wards, is emphysematous gangrene, or, in modern parlance, malignant œdema (Koch).

The general adaptation of the great principle of cleanliness, in even insignificant lesions and operative interference, has almost done away with this formidable foe to the wounded, and where it does appear it follows not, as a rule, in the wake of the surgeon's instruments. In our days, if at all, it is met with in open fractures and deep lacerated wounds which in some manner have come in contact with filthy material harboring a certain pathogenous germ.

It is at present a well-settled fact that this form of wound-infection owes its origin to a well-characterized bacterium, the bacillus of malignant œdema.

This is not the place to exhaustively discuss the form, life-properties, habitat, and manner of growth of the bacterium in question. Suffice it to say that it was first experimented with by Pasteur, who obtained it by injecting putrid blood into animals. They died, and Pasteur called the bacillus invariably found in the tissues affected *vibrion septicque*. Koch afterward re-discovered it by introducing garden-earth into pockets made under the skin of mice, rabbits, etc. He called the micro-organism found after the death of the animals, the bacillus of malignant œdema. It is most abundantly met with in the neighboring tissue of the skin-pocket and on the serous membranes of the abdominal and thoracic cavities.

The pathological changes produced by the microbe by means of hypodermic injections of fluid containing the germ,

in animals, are diffuse œdema of the neighborhood of the inoculation, accompanied by emphysema. The latter is due to gas-formation in the tissues. The disease (in animals) clinically resembles anthrax, from which, however, it can be easily differentiated by the manner of death, by the form of the bacillus found, by its manner of spreading through the organism (the anthrax bacillus proliferating in the blood, the other in the tissues), and, last, by its behavior in the cultivating media.

Leaving all the other distinguishing characteristics aside, I will only state that our bacillus, when examined under the microscope, is a slender rod, growing at times into false threads, resembling very much the anthrax bacillus, from which it differs by the rounded ends, the latter having both of its extremities slightly excavated.

It is met with almost everywhere, to wit: in the superficial strata of manured ground, in putrefying cadavers, in old rags, in dish-water, etc. It forms spores, and it is generally in this state that the bacillus occurs in the substances named and is introduced into the living body.

The artificial production of malignant œdema in man has been observed before. Thus Brieger and Ehrlich¹ have described it in two cases of typhoid fever following the subcutaneous injection of tincture of musk, which probably contained the organisms. The patients died. I myself² reported a fatal case of malignant œdema in a woman who had been in the habit of producing abortions on herself and had probably employed an unclean instrument in the last operation for that purpose.

Two recently observed cases following the use of the hypodermic needle in morphinists are perhaps of interest to those who have frequent dealings with such patients.

That the morphine-injector is very careless as to the condition of his syringe, and that he will continue its use, although every injection is sure to be followed by an abscess, is a fact too well known to be dwelled upon. That in such

¹ Berlin, klin. Wochschr., 1882, No. 44.

² Bremer on Malignant Œdema and Fat Embolism. American Journal of Med. Sc., June, 1888.

cases the syringe is "infected" with pus-producing organisms, is well known. To what extent the weakened general condition, the lowered tone of the system, and the inadequate or vitiated metabolism³ of the body, favor the propagation of these pus-producers when once introduced into the tissues is as yet undecided. We know that even without special preparation of the "soil," the pyogenic staphylococci, when introduced into animal tissues, will set up typical suppuration. They differ in this respect from the bacillus of malignant œdema, of which the human body, as a rule, is not susceptible. But that under certain conditions this bacillus may invade the human organism, the following two cases well illustrate:

CASES.

Case I.—Mrs. M., aged thirty, has been the victim of the morphine habit for a number of years; she has also been addicted to the use of stimulants to a considerable extent. She is stout in appearance, but her flesh is soft and flabby. The abdominal walls are especially thick with a large amount of adipose tissue. She has been in the habit of using the hypodermic syringe (fifteen grains of morphine a day), and her arms and abdomen are tattooed with the marks of the needle. One of these punctures had inflamed, and when seen by Dr. Prewitt, of this city, she had high fever, frequent pulse, some irregularities of the heart's action, anorexia, and diarrhœa. Over the abdomen, a little above the level of the umbilicus and between that and the left lumbar region, there was a broad inflamed indurated patch, four or five inches in breadth and five or six in length, which fluctuated in the centre and was resonant on percussion over this part. A thought of an inflamed hernia, with a flatulent knuckle of bowel included, suggested itself; but the history of the case excluded this. Ether was administered at the solicitation of the patient. Upon making an incision, the bistoury entered as though penetrating a bag of air, giving exit to a puff of exceedingly fetid gas, followed by a quantity of equally fetid pus.

For a moment the possibility of having cut into a knuckle of bowel caused some trepidation, but a slight investigation showed that the gas came only from the abscess-cavity.

Unfortunately none of the pus was preserved for examination. Judging the case in the light of recent experiences,

³ Compare the furunculosis in diabetes.

our present knowledge of gas-containing abscesses, there is no doubt that the bacillus of malignant œdema would have been found. (Compare the next case.)

The probable carrier of the infectious material was the syringe, or perhaps the water in which she dissolved the morphine. As she injected it secretly, she probably was not very particular in choosing the purest article, and had, at times, to take any kind of water at hand.

The incision was made free, and under thorough antiseptic treatment the abscess assumed a healthy character and rapidly filled up. A second abscess formed in the lower border of the indurated area of much smaller dimensions, and was opened, but presented none of the peculiar features of the first.

The further progress of the case and its treatment are not germane to the subject of this paper, and will not be dealt with here. I saw the case only after healthy granulations of the wound had set in, when none of the infectious material could be obtained for examination.

Case II.—M. C., aged twenty-eight, has been injecting morphine for the last eight years. The habit has lately grown upon him to such an extent that he has used up to thirty grains a day. In order to prevent fainting-spells, as he alleges, and to be competent to discharge his duties as a bank-officer, he was obliged to inject with the utmost quickness, and since the skin of his arms was nearly unfit for further injections, owing to the extensive scars, the results of former suppurations, he had formed the habit of injecting through his pantaloons into the muscles of the thigh and the nates. He claims that he could do this in the bank, unnoticed by the other employés. At the instance of his family he had resolved to enter upon a "weaning cure," and the day for its commencement had been set. Probably to once more enjoy the effects of the drug to the fullest extent before parting with it "forever," he injected for a few days more recklessly than ever, and presented himself on the morning of the day appointed for the beginning of the ordeal in a most wretched condition. On examination an extensive reddish-blue swelling, fully the size of two fists, involving almost the whole of the right buttock, which had been of late the favorite place of the injections, presented itself. There were also painless nodules, representing abscesses, all over the body, varying between the size of a bean and that of a walnut. There was only a slight elevation of temperature, pulse 90, sickness of the stomach, and general malaise.

A free incision having been made, an enormous amount of stinking, bubbling, pale reddish looking fluid, mixed with white streaks, burst forth. Large shreds of mortified connective-tissue and liquid fat were also discharged. The peroxide of hydrogen was poured into the cavity twice a day, and the enormous defect, large enough to hold a man's fist, healed up in a remarkably short time.

The oxygen was resorted to from theoretical considerations. The bacillus of malignant œdema is an anaërobe; exposure to the atmospheric air, or, still better, oxygen in some form or other will interfere with its proliferation. Probably, however, the simple free opening of the abscess would have sufficed.

The culture-experiment (gelatine and agar) revealed the presence of the staphylococcus aureus and albus; the piston of the syringe was planted in nutrient gelatine, and on the third day the unmistakable gas-formation took place around the leather disks. No further inoculation-experiments were undertaken.

There was then a "mixed infection" in the sense of Brieger and Ehrlich.⁴ The bacillus of malignant œdema is one of those microbes which, in man, are comparatively harmless. It takes another microbial invasion to aid the virus under consideration to gain a foothold in the tissues of the body. Experiments carried on lately in this direction have proved that quite a number of micro-organisms, which are absolutely innocuous when introduced singly, become pathogenic when they enter into a higher organized body in combination with another equally harmless microbe.

In the two cases reported, and in others similar in kind, it seems that the pus-producers pave the way for the successful colonization of the bacillus of malignant œdema.

Another factor which probably contributes to the greater facility of the microbe establishing itself in the body is to be found in the manner of inoculation. Experiments with a number of germs have proved the fact that inoculations are negative when made by the knife, *e. g.*, making an ordinary pouch under the skin; whereas the inoculation proves successful when done by the hypodermic syringe (Sputmann-septicæmia in rabbits). It is perhaps unnecessary to call to mind the many mishaps which were caused by the syringe during the late Elixir of Life craze.

⁴ The abnormal metabolism as a predisposing agent has been alluded to above. Cf. loc. cit.

Periscope.

By LOUISE FISKE-BRYSON, M.D., CHAS. HENRY BROWN, M.D.

INSANITY AFTER ACUTE SURGICAL OR MEDICAL AFFECTIONS.

In the "University Medical Magazine," Dec., 1889, Dr. H. C. Wood has an interesting paper treating of this condition, concerning which it is very important that correct ideas be disseminated among the rank and file of the profession, as cases of this kind usually come under the observation of the family doctor, who sometimes fails to comprehend the situation. Though insanity following acute disease varies greatly in symptomatology, there is generally one common fundamental brain condition; and this fundamental brain condition bears no specific relation to the disease which has produced it, but may be the outcome of an altered nutrition produced by exanthematous disease, diathetic disorder, traumatism, or by a surgical operation. There are ætiological and symptomatic reasons for believing that insanities from these causes are identical in their nature.

Ætiological.—After disease, the symptoms develop during convalescence, when the specific poison has exhausted itself. Fevers, traumatism, surgical operation, that are followed by insanity, have one influence in common, *i. e.*, they all tend to exhaust or impair the nutrition of the nerve-centres. It is known that impairment of the nutrition of these centres, by lack of food, combined with anxiety, is capable of causing symptoms similar to those which are present in insanities developed after disease.

Symptomatic.—Though the cases vary much in detail, the general scope of symptoms and the general course of the disorder are identical. 1. There is always mental confusion, a mixture of excitement and mental power. 2. The cases nearly always end in complete recovery, if free from organic disease.

Confusional insanity, a term without pathological import, but expressing a necessary outcome of the pathological state, is probably as good a name as can be found for this condition. In chronic diseases, accompanied by great bodily and mental exhaustion—consumption, gout, rheumatism, after shipwreck followed by exposure and starvation—

the brain-tissue gradually passes into a condition of perverted and exhausted nutrition similar to that of confusional insanity. This form of alienation follows typhoid fever not infrequently, and probably constitutes the bulk of cases named puerperal insanity. To it, also, belongs the so-called surgical insanity. Within the last year the author has seen it develop after ovariectomy, perineorrhaphy, and after the removal of the breast for cancer. It may be due to emotional strain accompanied by exhausting circumstances. The mental enfeeblement following exhausting disease is very slowly recovered from—two years elapsing before a student, who had had typhoid fever, regained power of acquiring knowledge and doing high intellectual work. When confusional insanity is fully developed, there is almost invariably a general lack of nerve-tone—as shown by feeble circulation and coldness of the extremities—by general muscular relaxation, and failure of digestive power. The tongue is often so heavily coated and the breath so foul that the inexperienced may think digestive disorder the basis of the attack. The temperature varies in different cases, normal, low paroxysms of subnormal temperature. On the other hand, there may be distinct febrile reaction, especially in puerperal cases. When fever exists at all, the temperature-range is remarkable for its irregularity and extent, extreme pyrexia being often followed by sudden and marked fall below the normal. Mental symptoms seem to be contradictory. Many are those commonly supposed to be the outcome of paralysis of cerebral function, while others are sometimes considered evidence of excited, though perverted, cerebral activity. Mental confusion is the most characteristic manifestation of the disease. This expresses itself by inability to carry on a clear, connected conversation and to recognize familiar persons and places. The weakened judgment cannot distinguish between the contending claims of subjective and objective sensations. Realities and imagination are intermingled in hopeless confusion. Memory is pronouncedly affected, and numerous vivid hallucinations affect all the senses. A peculiar hallucination, so often present that it may be considered characteristic, is that *another person or persons are in bed with the patient*. (In the case cited of Mr. J., who recovered, this: "In his delirium, always desiring to be taken home; constant vagary, that there are babies in the bed who are liable to be hurt.") The delirium is commonly mild and non-aggressive. It may take on an active form, or the patient may be habitually quiet, but subject to paroxysms resembling those of acute mania. Any form of mania may be

counterfeited. Commonly there is fear underlying the aggressiveness and violence, resembling the fear of delirium tremens. When tremulousness exists, the likeness is very pronounced. The author thinks delirium tremens may be considered a form or variety of confusional insanity. The kaleidoscopic character of the hallucinations, the stupor, the peculiar mental state, the lack of dominant emotional excitement, of good bodily nutrition and of general nerve-force, together with the previous history of exhausting disease, traumatism, or emotion, should make the diagnosis easy.

Treatment.—First, maintenance of bodily warmth, by over-heated rooms, hot-water beds or bags, by the warmest covering whenever the bodily temperature is sub-normal. Rest, massage, and exercise are hygienic measures that must be applied in every case. To increase the nutrition of the nerve-centres and general tissues of the body, iron combined with bitter tonics, in small or large quantities, strychnine given in small doses to the limit of physical tolerance, and phosphorus continuously in small doses ($\frac{1}{120}$ to $\frac{1}{100}$ of a grain) are of great service. To obtain sleep or quiet delirious excitement, the bromides suggest themselves. These are powerful depressants to functional and nutritive activity of nerve-cells, and great harm has often been done by their free use in confusional insanity. Opium, hyoscine, chloral, and sulphonal are of value, the selection of a hypnotic being based chiefly upon the results of experiment in individual cases. As a calmative, the hot-pack is serviceable, and very active delirium has been apparently much benefited by free blistering of the scalp.

VERATRUM VIRIDE IN EXOPHTHALMIC GOÎTRE.

(*Ibid.*) Patient thirty-five, height above medium, weight ninety-three pounds; married, mother of three children; anæmic; greatly debilitated; heart apparently much dilated, without rhythm, with a wallowing movement; exophthalmia so great as almost preventing closure of lids; goître, not measured, but very prominent; mind deranged; duration of malady twelve years. A physician in a distant city, the first of many, recognized the disease, and advised the patient to return home at once, as she was liable to drop dead any moment. This was confirmed by Dr. W. D. Hutchins, of Madison, Ind., who ordered tr. veratrum viride three drops, morning and night, to be gradually increased to the utmost limit of tolerance. Three drops were barely tolerated at first; four drops produced such weakness that the patient was obliged to stay in bed for a short time.

Later, twelve drops were taken, night and morning, without inconvenience, this dose being continued twice daily for a year, then dropped to one dose daily for a few following months. Improvement was gradual but progressive. At the expiration of twelve months from the beginning of treatment, the goître, exophthalmia, and the mental symptoms had disappeared. Bodily weight was then 160 pounds. There has been no relapse.

Liegeois ("London Medical Recorder," August 10, 1889) speaks highly of *veratrum viride* in palpitation of the heart, due to various causes, especially when of functional origin. In palpitation associated with hypertrophy from valvular or peripheral lesions, it gives good results. Indications for the drug are the following:

1. Functional palpitation and arrhythmia, accompanied by heightened arterial pressure.
2. In palpitation and arrhythmia due to organic valvular lesions during the period of hypersystole.
3. In palpitation and arrhythmia during the first of the so-called arterial or vascular heart-disease.

Veratrine is irritating and even emeto-cathartic; *veratrum viride* has none of these distressing symptoms, and, like *strophanthus*, may be given for weeks together, if sufficient interval be allowed to elapse between each dose. If the dose be not excessive, no cumulative effect is produced and tolerance is not established.

HYPERPYREXIA IN HYSTERIA.

The "Therapeutic Gazette," of Dec. 16, 1889, calls attention to a notice upon this subject, that appeared earlier in a Danish journal, thence assumed a German dress, and then found a place in the "Lancet" of Oct. 12, 1889. The patient, after an attack of hæmoptysis, suffered from severe dyspnœa, cyanosis, and temporary asphyxia, several times during the night; after the attack there was loss of consciousness for some time, anxiety, and inclination to hallucinations. During the next two months the symptoms were repeated without any signs of phthisis. There was retention of urine. The temperature varied for three days from 103° to 104° F.; on the fourth day, at noon, it rose to 113°. The physician, Lorentzen, found the patient with other signs of inflammation; slightly delirious; temperature, one hour later, 108°; in the evening, 106.3°. The next day it rose to 113°, but fell in an hour to 99.5°. It varied in the next few days from 101.3° to 103.1°, and then became normal. Lorentzen considers the rise of temperature and respiratory disturbance as purely neuropathic. The aston-

ishing statement is made that Teale found the temperature in a hysterical patient 122° . Wunderlich records a case where it was 109.4° .

TRAUMATIC HYSTERIA.

The "Gazette des Hôpitaux," for Nov. 23, 1889, contains an admirable paper with the above title, by M.E. Brissaud. Hysteria he considers one and indivisible, whatever the cause that fans it into flame, whether traumatism, intoxication, lightning, terror, or even misplaced love itself. None of these are more than exciting causes, quite incapable of creating distinct forms of hysteria, and occupying, in the nature of things, a subordinate position. Accidents and profound emotions render certain persons hysterical, because they have an hysterical make-up; but the accident or the violent emotion cannot of itself create hysteria. Otherwise almost every one would be a victim, for some accident happens to even the best of us during the course of a lifetime; and it is well known that the men and women of keenest intellect and greatest usefulness are capable of the most profound emotion. "Passions are the most truly creating things in the world, and the want of passion the truest death or unmaking of things." Thus, in the normally constituted human being, profound emotion brings about power, and controlled passion liberty and happiness. Not so with abnormal humans, whether men or women. Emotion or an injury conquers them; and the result may be disturbances of sensation, hemianæsthesia, pharyngeal anæsthesia, disseminated hyperæsthesia, concentric restriction of the field of vision, dyschromatopsia, various paralyses, loss of muscular sense and the power of speech, contractures, laryngeal or œsophageal spasm, nervous crises and physical symptoms,—in a word, manifestations of hysteria. These occur in prisoners, in street-vagrants, in idle, luxurious women, in self-indulgent men, in the overworked of both sexes, in children sometimes, but only among the *hysterical*.

The subject of injury, poison, overwork, or emotion reacts according to its individual mechanism. An exciting cause only is necessary for such reaction. In this resides the specific nature of hysteria, for it is in itself almost equal to a function, responding to external stimuli. The retina has but one method of reaction to irritations, however they may be produced, whether by pricking, burning, pinching, faradization, etc. The reaction is always a visual sensation. This visual sensation is a physiological constant quantity, while hysteria is the pathological constant quantity. If

railway-spine or railway-brain presents the phenomenon of hysteria, in what way do they differ from hysteria proper? Neurasthenia may be added the other condition (hysteria), as it is frequently, but no hybrid disease results, any more than when exophthalmic goitre and hysteria co-exist, or Addison's disease, or migraine. The two may be combined, but never compounded. Vibert says there is no special form of general paralysis following an injury. The disease acts just as in spontaneous cases. Charcot makes the same statement in regard to hysteria. The author objects seriously to so-called toxic hysterias, for the nervous symptoms in cases of poisoning may express themselves in three distinct pathogenic forms or stages of special evolution, that may or may not be hysteria. In the first, the nervous symptoms disappear simultaneously with the general phenomena of intoxication. They owe their existence to the presence of some toxic element, and hysteria is not their source. In other cases, as the poison is eliminated, nervous manifestations increase; but alterations in sensibility always affect circumscribed territories under such circumstances, as the region of a plexus, a nerve-trunk, or its branch. Paralysis of motility is accompanied by muscular atrophy sometimes, and the reaction of degeneration always. In a third order the patient has recovered long ago; the poison has been altogether eliminated; but the man or woman is paralyzed, anæsthetic, with muscular, articular, and cutaneous sensibility deficient or lost and faradic reaction normal, and other symptoms characteristic of true hysteria. This is simply the history of an hysterical person who has been poisoned, and not the history of any special new disease.

HYDROTHERAPY IN MENTAL DISEASE.

In the "*Bulletin de la Société de Médecine mentale de Belgique*" (Dec., 1889) forty-four pages are devoted to a study of this subject, by Dr. Jul. Morel. Various forms of application for different abnormal states—as neurasthenia, epileptic, choreic, or hysterical mania, hysterio-epilepsy, melancholia, general paresis, etc.—are considered with care and precision. The bibliographical index is of value, comprising some twenty-eight sources of information. Mechanical methods of cure have not as yet received all the attention they deserve, it being easier to take something than to do something. In the treatment of mental and nervous disease there is, without doubt, a brilliant future for hydrotherapy.

L. F.-B.

GASTRIC NEURASTHENIA AND ITS TREATMENT.

Prof. Dujardin-Beaumetz, according to the translation of E. P. Hurd, M.D. ("Therapeutic Gazette," Jan. 15, 1890), after describing a clinical case with marked dilatation of stomach, slight enlargement of the liver, and an easily manipulated and mobile left kidney, with a history that goes back to a distant date of disordered functions of the digestive tube, speaks of this class of neurasthenias who have dilated stomachs, as numerous, and comprises, in the expressive phraseology of Jrastour, the "*déséquilibrés du ventre*" ("those whose stomachs are not equilibrated"), and that they constitute the rank and file of the neurasthenics. The author discusses three theories, which have been broached to explain these disorders dependent on bad functioning of the nervous system.

The nervous theory—so ably defended by Beard—lately defended by Leven, who assigned modifications of nerve-cells of the solar plexus a preponderant rôle: "This needs an anatomical demonstration more precise than has been furnished by Leven." The author also asks: "What causes these solar-plexus irritations?"

The second theory: a more plausible one advanced by Glenard, which is, "that all sorts of functional nervous derangements included under the head of neurasthenia depend on a modification effected in the reciprocal relations of the different portions of the intestines"—displacements to which is given the name of *ptoses*, and may pertain to all the organs contained in the abdomen.

Splanchnoptosis (terminology of the Lyons school) is seen most frequently in prolapsus of the right flexure of the colon and first part of the transverse colon.

This theory of a fundamental organic lesion of *enteroptosis* was first made known by Glenard in 1885, and confirmed by many others since. The mobility of the kidneys—nephrotosis—is dependent, mechanically, upon the enteroptosis. To oppose this is the humoral theory of Bouchard—auto-intoxications (from ptomaines, leucomaines, etc.) of intestinal origin—which causes congestion of liver—nephrotosis gastrectasis—and a long train of nervous phenomena, which are dependent upon the direct irritative action of ptomaines on all departments of the organism.

After comparing these two theories of Glenard and Bouchard, and the inability which either possessed to explain all the pathological changes that occurred in true gastric neurasthenia—such as dilatation of stomach and enfeeblement of gastric mucoid, which to a great extent must go

back to congenital causes—he regarded that Bouchard's theory deserved the most acceptance, and considered the pathological condition of the large intestine to vary—sometimes dilatation of the descending colon—and some patients, with all the phenomena of gastric neurasthenia, to have, on careful examination, dilatation of the entire colon, and not the stomach, and to be treated, not by lavage of the stomach, but by antiseptic lavage of colon and rectum.

The author next considered the treatment of Glénard, summarized as follows:

1. To combat the visceral prolapsus and augment the abdominal tension by means of an appropriate abdominal belt.

2. To regulate the intestinal evacuations.

3. To institute a special regimen, in order to strengthen the digestive organs.

The author then considered briefly the importance of distinguishing between dilatations of stomach and intestine; the most important sign being *bruit de clapotement*, which must be heard below an oblique line which passes from borders of the free ribs and the umbilicus. To obtain this *bruit* it is often necessary to resort to overcoming the vacuity of the stomach by having the patient drink a glass of water, and the effect of contraction of the abdominal recti muscles by patient taking a full inspiration and then press suddenly upon the abdominal wall, grasping with both hands the two sides of the abdomen, etc. Examination in iliac fossa will develop the *bruit* in intestinal dilatation.

Summary of treatment for gastric neurasthenia is as follows:

- “1. The patient may take with each meal five grains each of salicylate of bismuth, magnesia, and bicarbonate of sodium in capsule.

- “2. He may take, on going to bed, a dessert-spoonful of the compound licorice-powder.

- “3. Every day he may have a cold-jet douche, applied along the vertebral column. The duration of the douche should not exceed fifteen seconds (if the patient be a lady, douche the feet with warm water); energetic dry friction with a flesh-brush after the douche.

- “4. Walks in the open air, muscular exercises (opposition gymnastics, fencing, etc.) are beneficial.

- “5. Pursue with rigorousness the following dietetic regimen: Let there be seven hours at least between the two principal meals. If the patient takes three meals a day, the first should be had at 7.30 A. M., the second at 11 A. M., the

third at 7.30 P. M. If two meals only should be eaten, let the first be at 10 A. M. and the second at 7 P. M. Never to eat or drink between meals.

"Let the diet consist largely of eggs, cereals, starchy foods generally, green vegetables, and fruits.

"*a.* The eggs to be but little cooked (creams, custards).

"*b.* The starchy foods to be thoroughly cooked (mashed potatoes, stewed beans, lentils, revalrescière, racahout, lactated farina, panada, rice in all its forms, macaroni, biscuits, buns, hominy, oatmeal, etc.).

"*c.* The vegetables should also be well cooked (boiled, mashed carrots, turnips, peas, cooked salads, spinach, etc.).

"*d.* The fruits should be stewed, with the exception of strawberries and grapes.

"Use toasted bread instead of plain bread. Eschew from the dietary, game, fish, mollusks, crustaceans, old cheese, as well as liquid foods, and soups that are too thin.

"To be permitted: soups that have been thickened, gruels of various cereals, wheat, rice, Indian corn, etc.

"As for drinks, take only a tumblerful and a half of a mixture of light white wine with ordinary water or Alet water; no gaseous waters; no pure wine; no whiskey or other distilled liquid.

"You see the important part played by diet, and especially the vegetarian diet, in the treatment of gastrectasia."

CRIMINAL RESPONSIBILITY IN NARCOMANIA. By Norman Kerr, M.D., F.L.S. ("Medico-Legal Journal," Dec., 1889).

"In insanity it is now generally conceded that there is a lesion of the brain, though this cannot always be detected on a post-mortem examination. There is now as much evidence to show that there is a brain lesion in inebriety, that diseased condition which I have ventured to call narcomania (a mania for intoxication by any anæsthetic narcotic). In acute mania, as in delirium tremens, this lesion is usually quickly repaired. In some forms of mental unsoundness and of narcomania, this lesion is so persistent that a prolonged course of treatment is required, while in a sensible proportion of cases the lesion is practically irreparable.

"In the interests of justice as well as in fairness to the accused, in all cases of alleged criminal offences committed either while under the influence of an alcoholic or other anæsthetic, or by a known inebriate in a non-narcotic interval, there ought to be a skilled inquiry into the previous health-history and heredity of the panel at the bar."

A CONTRIBUTION TO THE STUDY OF EXOPHTHALMIC GOITRE. By Græme M. Hammond, M.D. ("New York Med. Jour.," Jan. 25, 1890).

After drawing attention to the fact of there having been very little advancement of original research in the pathology of the disease, the author drew especial attention to a symptom which deserves confirmation from others. This he called, from its first observer's name, "Dr. Louis Bryson's symptom," which consists in the inability of the patient under forced inspiration to expand the chest up to the normal extent. In every case examined this was confirmed. In some the loss of power to expand the chest was remarkable, and according to Dr. Bryson, when the expansion is reduced to half an inch or less, the termination of the case is invariably fatal, and was substantiated by Dr. Hammond in one case. In eight cases that recovered, the power of the chest-expansion was restored.

He refutes the theory that disease of the cervical sympathetic causes the disease, and considered the theory of a central lesion "to a circumscribed lesion affecting the vagus nucleus, vaso-motor nucleus, and the respiratory nucleus" more tenable, and supported by the case of Dr. W. Hale White, who "reported a case where the sympathetic was found to be healthy. A series of sections were made from the lowest part of the medulla to the corpora quadrigemina. At the level of the lowest part of the olivary nucleus there was, just under the posterior surface of the medulla, evidence of slight inflammation. The next few sections were quite healthy, but those in the neighborhood of the sixth nerve showed considerable changes. Immediately under the posterior surface of the medulla, extending from the mesial line as far out as the restiform bodies, which were slightly implicated, were numerous hæmorrhages. The area occupied by these hæmorrhages did not extend deeply, so that, except for a slight implication of the nerve-cells of the sixth nucleus on one side, the nerve-cells had escaped injury. The hæmorrhages seemed almost entirely limited to the posterior part of the *formatio reticularis*, but there were two or three small, deep ones. They were not marked

at this level, but were observed up to the lower part of the aqueduct of Sylvius."

Dr. White believes this is the first case where organic lesions have been discovered in the medulla in exophthalmic goitre, but Lockhart Clark reports a case where the "corpora quadrigemina and the medulla, particularly on its posterior part, were very soft, and, on minute examination, displayed the usual appearance of common softening."

Fox states that "the weak point in this theory of central origin seems to be that there is so seldom any dilatation of other vessels besides the thyreoidal." There is a strong probability that there is a general dilatation of the blood-vessels. It has been conclusively shown that in exophthalmic goitre the electrical resistance of the patient is very much diminished below the normal point. And although as yet there is no absolute proof, it seems plausible and probable that a general dilated condition of the vessels would account for the greatly diminished electrical resistance.

In many instances no lesion has been discovered at all, and the burden of proof goes to show that exophthalmic goitre is frequently a reflex neurosis.

Those cases reported the reflex disturbances as situated within the nasal cavity. The author considered irritation in other parts of the body might do the same, perhaps more liable parts being the eyes, nasal cavity, and genito-urinary apparatus.

Dr. Hammond confined the discussion of the treatment principally upon the consideration of carbozotate of ammonium, the value of which in the treatment of exophthalmic goitre was first discovered by Dr. A. C. Combes, his clinical assistant, and finally says:

"I have used it on three cases of my own, with, I think, decidedly good results. Its use is, however, limited, and for reasons which I will now mention cannot be given indefinitely.

"Following the directions of Dr. Combes, I have given the remedy in pill-form (each pill containing one grain of the drug), three times a day for the first week. In the second week two pills three times a day are given, and, if it can be borne, three pills three times a day in the third week. The physiological effects of the drug are very decided. They were observed by Dr. Combes, and his observations have been verified by my own. At about the end of the first week the skin and conjunctivæ assume a slight saffron color, which deepens if the drug is persisted in. Then a peculiarly unpleasant odor emanates from the

body, which is identical with that produced by dirty feet, and can be distinctly noticed if you approach within six or eight feet of the patient. Following this, severe gastric disturbances show themselves. It is rarely possible that patients can take this remedy longer than three weeks, but while they take it the effects upon the heart, the respiratory tract, and the exophthalmia are undoubted."

A CASE OF CEREBRO-SPINAL MENINGITIS.

Dr. J. F. Erdmann, in the same number of the "New York Med. Jour.," reports a case of cerebro-spinal meningitis with remarkable diminution of number of respirations. On the seventh day the number of respirations had fallen to four in a minute. A hypodermic of one-fiftieth grain of sulphate of atropia was administered, and within three hours the average respirations per minute were nine. They were averaging six the next day; the following day thirteen. Sulphate of atropia was the drug used to combat this symptom. Case recovered.

"Leyden attributes the diminished and Cheyne-Stokes respirations observed in the late stages of this disease to pressure upon the medulla produced by œdema, basing his opinion on the observations of Schiff after the artificial induction of hæmorrhage in the vicinity of the medulla in dogs."

C. H. B.

ON ALIMENTARY THERAPY IN THE TREATMENT OF MENTAL DISEASES.

Lattier ("Ueber alimentäre Therapie bei Behandlung der Geisteskranken," in "Centralblatt für Nervenheilkunde, Psychiatrie und gerichtliche Psychopathologie," xii., 20, 1889) distinguishes, in the alimentation of patients suffering from mental diseases, those which are only cared for and those which are actively occupied, who need more nutritious food, which must contain a greater amount of nitrogenous constituents. The proportion for those who are only cared for he determines, according to Gasparin, 264 grammes of carbon and 12 grammes of nitrogen as sufficient; while for those patients who also work he recommends 309 grammes of carbon and 25 grammes of nitrogen.

He especially investigated the peptones. The peptonizing is caused through the action of the gastric juice and the secretion of the pancreas. The same property is possessed by *carix papaya*, which is frequently employed. In the nutrition of a man Lattier assumes 1 grm. of peptone to 1 kilogrm. of the body-weight, but considers the liquid peptones superior to the dry ones, because the former are

much more readily taken. They may be prepared in bouillon or wine if they have to be given by the œsophageal sound or per rectum.

200 grms. of dry peptone may be obtained from 1 kilogram. of meat. The following formula is recommended :

Malaga, - - - - - 25 grms.

Dry peptone, - - - - - 5 "

As regards the employment of raw meat, this was given with great result in the Quatre-Mères Institution, for the last fifteen years, in the following preparation :

Raw scraped meat, - - - - - 100 grms.

Powdered sugar, - - - - - 40 "

Wine, - - - - - 20 "

Tinct. cinnam., - - - - - 5 "

The mixing is done in a mortar with addition of the liquid. A pap is formed, which is readily taken by the patient if prepared on bread like a sandwich.

Lattier attaches great importance to the use of pulverized meat, which possesses the same nutritive properties as bread with bouillon, although not the same as raw meat, as was shown by the experiments on animals. It may be administered once or several times a day, in doses from 50 grms. to 300 grms. To facilitate the ingestion and prevent the remaining of it in the mouth, the following formula is recommended :

Potato-flour (*poudre*), - - - - - 5 grms.

Pulverized meat, - - - - - 50-100 "

Water or bouillon, - - - - - 200-400 "

Salt or tinct. of cloves, - - - - - q. s.

The whole mixture is boiled, with continuous stirring ; the semi-fluid preparation is easily taken.

Kefir is considered superior to koumyss, because the former possesses an agreeable taste and really nutritive properties. Its preparation takes place by the action of a peculiar mushroom, which grows in the Caucasus, and which causes spirituous fermentation. It is a somewhat thick liquid, of a milk-color, and of a somewhat acid and peculiar taste. The chemical combinations of milk, kefir, and koumyss are given comparatively :

	MILK.	KEFIR.	KOUMYSS.
	(Sp. gr. 10.28.) (2 days old.)		
1st contains albuminoid constit., -	48	38	11.2
2d " fat, - - - - -	38	20	20.5
3d " water and salts, - -	873	905	918.3
4th " alcohol, - - - - -	—	8	16
5th " lactic acid, - - - - -	—	9	11.5
6th " lactose (milk-sugar), -	41	20	22

According to the above table, kefir contains much more albumen than koumyss, less alcohol, and has a less harsh taste. Lattier thinks that koumyss as a nutriment contains too much alcohol.

The varieties of milk set in fermentation are especially appropriate for alcohol-drinkers, as they elevate the tonic and digestive action of the milk by their containing alcohol and lactic acid. They have shown themselves to be very efficacious as medicaments and nutriments.

In the nutrition of insane patients who defy the taking of food and are to be fed through the œsophageal sound, it must be remembered that the supply should be a more abundant one than in sane patients, because the tissue-waste is a more extensive one. Lattier found this confirmed by chemical experiments, especially in patients who were suffering from melancholia activa. Hence are to be especially recommended fats, as they diminish the waste of albumen, in combination with eggs, meat, bouillon, etc. He recommends for feeding with the sound the following formula :

	NITROGEN.	CARBON.
4 eggs, weighing 216 grms., -	4.11 grms.	32.40 grms.
2 litres of milk, - - - -	13.20 "	160 "
Bordeaux wine, 215 grms., -	0.04 "	10 "
Pulverized meat, - - - -	3.91 "	14.32 "
	<hr/>	<hr/>
	21.26 "	216.72 "

The nutrition by peptonic injections, per rectum, of those patients with whom the introduction of the œsophageal sound is impossible, is especially considered and thought very well serving the purpose. Although they cannot be continued for any length of time, they are yet of great value for a certain time. Before the application of the same an ordinary injection is recommended, to which a few drops of tinct. opii are added in order to weaken the contractions of the rectum. Lattier recommends, according to his own experience, the following formula :

Dry peptone, - - - -	3 teaspoonfuls.
Yolk of egg, - - - -	1.
Milk, - - - -	125 grms.
Tinct. opii, - - - -	5 drops.
Pulver. amidon. - - - -	5 grms.

The addition of amidon is said to render it more easily.
P. and P. (Boston).

Society Reports.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, November 25, 1889.

The Vice-President, Dr. WHARTON SINKLER, in the chair.

Dr. CLARKE, resident physician at the Philadelphia Hospital, presented to the Society a patient suffering from

MERCURIAL TREMOR.

The patient was in Dr. Dercum's wards, temporarily in charge of Dr. Mills. The following are the notes of the case from the records of the hospital.

J. K., aged eighteen, single, had been working for a large Philadelphia hat manufacturer for two years and a half. He handled the "fur" used in making felt, and worked in an atmosphere full of the dust from this fur. He was well until seven months ago, when a tremor developed in his hands. This has increased and extended to his legs, tongue and lips. For this "nervousness" he went to the hospital, December 19, 1889.

His intellect seems good, although he states that his memory has failed in the last few months. He has no pain or headache, sleeps well, and does not seem emotional. His powers of co-ordination are unimpaired, and he has no paralysis. He is fairly well nourished, but anæmic and weaker than he was some months ago. The dynamometer records sixty-five for each hand. The legs are somewhat spastic, or perhaps exhibit a pseudo-spasticity. Knee-jerk is present in both limbs, but sometimes seems to be absent because of the forced position of flexion in which the legs are held. There is no true ankle clonus, but examination for clonus throws the limbs into violent vibration.

The tremor affects all parts of the body. It is most marked in the hands where it first started. When he holds the arm extended the whole limb is affected by a general vibration, most positive at the wrist. The hand tends to assume a slight extension at the wrist, and a slight flexion at the metacarpo-phalangeal articulation. There is a tremor of the head and trunk and also of the tongue. The articu-

laris of the eye is thrown into irregular spasm during the examination of that organ. The tremor is coarse, affecting different parts of the body coincidentally, but in a different degree; it is increased on voluntary motion; if the limbs are handled abruptly the tremor is so much increased as to resemble a case of paramyoclonus. At times he has the appearance of a person in a severe fit of the ague.

At some of the examinations made of this patient there appeared to be losses of sensation; with the æthesiometer he was unable to distinguish two points in the forehead at two centimetres distance; he did not complain of quite severe prickings with a needle. His speech is of a tremulous, slightly feeble character. His gait is not much affected. His pupils respond to the light, and the special senses are not impaired. His pulse is usually about 72, and there is no palpitation of the heart.

Three months after the beginning of the tremor, his gums became red and sore, but there was no increase of the flow of saliva, and his teeth were not loose. He had neither diarrhœa nor constipation. After a time the redness of the gums disappeared.

Dr. MILLS spoke of the rarity, in his own experience at least, of mercurial tremor. He also discussed the peculiarities of the tremor as exhibited by this patient. In the wards for nervous diseases of the Philadelphia Hospital, he had at the same time, besides this case of mercurial tremor, patients exhibiting the tremulous phenomena of alcohol and lead poisoning, of paralysis agitans, disseminated sclerosis, and parietic dementia, as well as cases of ordinary chorea, Huntingdon's chorea, and paramyoclonus. The tremor seemed to resemble most that present in some cases of disseminated sclerosis, although the patient's tremulous, enfeebled speech was perhaps more like that observed in dementia paralytica. On exciting or handling the patient the trembling increased and spread, much as in reported cases of paramyoclonus. Gowers says that the symptoms often resemble those of general paralysis of the insane more than any other disease, but the preponderant tremor, inequality of pupil, optimism, and indications of spinal degeneration are absent.

Gowers gives the following description of mercurial tremor which, it will be seen, closely corresponds with the account of this patient:

"A peculiar tremor, known among workmen as the 'trembles,' and medically as 'mercurial tremor,' is the most common and characteristic symptom. It is at first occa-

sional, occurring only when the patient is excited, and it is always increased by emotion. It usually begins in the face and tongue, and then invades the arms, and afterwards the legs. At first the tremor occurs only on movement, but ultimately it may become constant. In the former case the condition of the patient resembles that of one suffering from disseminated sclerosis, except that the tremor is less wide and less irregular than in characteristic cases of the latter disease. When constant, the tremor resembles that of paralysis agitans. During sleep the tremor usually ceases, but in extreme cases may only lessen. It interferes much with articulation, rendering the speech stammering and hesitating. When considerable, it may render the movement of the arms so unsteady that the patient cannot feed himself, and his gait becomes affected. At first the limbs are strong, but after a time muscular power is impaired, sometimes more in one limb than in another, but it rarely progresses to complete loss. Reflex action and power over the sphincters are always impaired, and electric irritability of the muscles is normal throughout.

"Psychical symptoms are also common, and may precede the tremor. They have been very carefully studied by Kussmaul. Irritability and a difficulty in giving attention to a subject are often the first symptoms, and may be accompanied by considerable mental distress and sleeplessness. Hallucinations sometimes occur, and there may even be outbreaks of maniacal excitement, but the insanity rarely corresponds to any distinct variety. This condition has been termed 'mercurial erythism.' It is sometimes accompanied by headache and palpitation of the heart.

"Sensory symptoms are present in many cases; pains, especially in the region of the fifth nerve and in the joints; formication in the limbs, and even loss of sensibility to pain; paroxysms of distressing sensations in the thorax, resembling those of asthma. The sensory disturbances always augment the mental instability. In rare cases more grave cerebral symptoms occur—considerable hemiplegic weakness, aphasia and deafness."

Dr. GEORGE E. DE SCHWEINITZ.—Mercury manifests poisonous symptoms independently of acute toxæmia the result of injection of a soluble salt, by the appearance of the ordinary stomatitis (ptyalism); in tremor and allied nervous manifestations; or as "mercurial cachexia." Dr. H. C. Wood, in his lectures in the University of Pennsylvania, has long taught that if the metal is introduced by inhalation, tremor or similar phenomena result; if by the

mouth or through inunction, stomatitis; and, in the absence the elimination through iodide of potash, "mercurial cachexia."

Stated Meeting, December 23, 1889.

Dr. JAMES HENDRIE LLOYD in the chair.

Dr. ROSS R. BUNTING read a paper on "Paralysis of the Serratus Magnus." (See page 67.)

DISCUSSION.

Dr. CHARLES K. MILLS.—I remember only one case of this kind, and that was seen at the University Hospital Dispensary some years ago. The diagnosis of paralysis of the serratus magnus muscle is not difficult if the paralysis be limited to that muscle and the case is carefully studied. Several conditions at first sight might be mistaken for this paralysis. The appearance of the scapula is not unlike that seen in some cases of rotatory lateral curvature of the spine. The test in such cases is of course to have the patient attempt to perform the physiological actions of the muscle. I have recently seen two cases in which the appearance of the shoulder when at rest was similar to that shown in the photographs. One was a case of rotatory lateral curvature; the other was a case of considerable lop-sidedness, with some curvature and wasting of muscles. It is stated that dislocation of the fibres of the latissimus dorsi passing over the point of the scapula sometimes occurs and causes a somewhat similar deformity.

The next paper presented was by Dr. M. IMOGENE BASSETTE on "Two Cases of Paralysis occurring during the Puerperal State." (See page 93.)

DISCUSSION.

Dr. FRANCIS X. DERCUM.—I entirely agree with Dr. Bassette in regard to the organic nature of the affection and its probable association with occlusion of the blood-vessels from some morbid change, septic or otherwise, in the blood. It is difficult, indeed, to form any other hypothesis for these cases.

Dr. CHARLES K. MILLS.—I should like to hear some discussion in regard to the cause of this condition in cases without heart-disease. The question arises whether or not

it might not occur from embolism as a result of phlebitis. In the first case reported to-night there were no acute symptoms, no pain, and no local symptoms. The second case might be of septic origin—the patient having had a sudden chill, followed by paralysis.

Dr. JAMES HENDRIE LLOYD.—I have studied this subject in the preparation of the paper referred to by Dr. Bassette, and I can see no reason why we should try to find a special name or erect a special pathology for these cases of organic hemiplegia following labor. Dr. Mills suggests the possibility of embolism. Hemiplegia occurring during the puerperium, as the result of embolism from affection of the heart, is simply a coincidence of the labor. The indications are that these cases reported by Dr. Bassette are organic, but whether they are embolic or hæmorrhagic it is difficult to say. We have not the grounds to differentiate between these two accidents. Whether or not they are due to special conditions of the puerperal state, such as sepsis, could only be determined by a study of the pathological conditions in this state. If the woman were septic, we might suppose that there was thrombosis of embolism.

Dr. H. A. TOMLINSON read a paper on a "Case of Acute Melancholia during the Progress of which there appeared 'Argyle-Robertson' Pupil, with Abolished Patellar Reflexes on One Side and much diminished on the Other."

DISCUSSION.

Dr. CHARLES K. MILLS.—The most remarkable features in this case were the presence and disappearance of the Argyle-Robertson pupil and the absence and reappearance of the knee-jerk. We have been in the habit of regarding these two symptoms as of vital importance in making a diagnosis of incurable mental affection. They are of particular importance in making the diagnosis of parietic dementia, whether it be of the spinal form or of the more ordinary type. The mental symptoms in the case reported are certainly not those of parietic dementia, although in parietic dementia there is often a long stage of depression, from which the patient may rally, to sink again.

Dr. J. MADISON TAYLOR.—In regard to the appearance and disappearance of the knee-jerk, I would say that we cannot now attach as much significance to this sign as did the older writers. In the recent researches made by Dr. Mitchell, and in which I largely assisted, great variations were found in the knee-jerk in presumably healthy indi-

viduals. In some individuals it would sometimes disappear, while in the same persons at other times it would be immensely increased. The variations in the knee-jerk would seem to have more to do with varying states of nerve wave or impulse or whatever it may be that varies in the economy.

Dr. JAMES HENDRIE LLOYD.—There are aberrant forms of general paresis where we do have prolonged melancholic states. I have now under my care a gentleman who for months has been in a state of melancholia, and who has had no true expansive delusions.

In reference to the knee-jerk, I may state that I have under observation a case of general paresis in which there is exaggerated reflex on one side and complete or almost complete abolition on the other.

Dr. GEORGE E. DE SCHWEINITZ.—I would ask whether in this case the fields of vision were taken.

Dr. TOMLINSON.—They were not.

Dr. DE SCHWEINITZ.—It is excessively difficult in these cases of narrow pupil, especially if there is a high error of refraction, and more particularly mixed astigmatism, as was the case in the patient reported, to determine whether or not there are changes in the optic nerve. I do not believe that the ophthalmoscope alone is always able to accomplish this. A careful study of the color-fields is necessary to ascertain slight changes in the papilla, especially if these exist in the deeper layer, the surface still being capillary.

Dr. CHARLES A. OLIVER.—It seems to me that the eye-symptoms in Dr. Tomlinson's case point very much toward general paresis in its early stages. I have seen quite a number of such cases at the State Hospital for the Insane at Norristown, where there has been more or less marked change in the optic nerve, as shown by the ophthalmoscope, and where, although it was impossible to get the fields of vision properly, yet it was determined in some cases that there were contracted fields. In these instances I have seen from time to time, at intervals of two to six weeks, marked changes in the size of the pupil, their relative shapes, and the degree of motility of the two irides. As these cases advance into the third stage of the disease, there is a marked lessening of the motor actions in absolute relationship with the amount of change in the optic nerve, objectively visible.

Dr. H. A. TOMLINSON.—I would merely state that Dr. Risley, who corrected the error of refraction, examined the eye-grounds carefully, and found no change.

Stated Meeting, January 27, 1890.

The President, Dr. H. C. WOOD, in the chair.

Dr. JAMES HENDRIE LLOYD exhibited a specimen showing anomalous distribution of the arteries forming the Circle of Willis.

Dr. JOHN B. DEEVER read a paper on "Trephining for Extra-Dural Hæmorrhage." (See page 83.)

Dr. J. L. BOWER read "Notes on Some Cases of Chorea and Tremor." (From Service of Dr. Mills at Philadelphia Hospital.)

Dr. CLARA ALEXANDER presented a specimen of "Leptomeningitis," with the following notes:

M. M., white, aged fifty-five, an ironworker, was a heavy drinker, but had no venereal history.

Two months before admission to the hospital he slipped and fell, striking his head on the curbstone. After this he complained of pain in one ear and never seemed to be quite well. He was spiritless, but continued his work. About one week before coming under observation he began to have frontal headache, the pain being very sharp. He had chills followed by fever, and pain across the front of the chest and stomach. After this there was no recurrence of the chill or fever. He had had a cough for about twenty years, always worse in Winter. There was no increase of the cough and expectoration after the occurrence of the chill, but the headache still continued, and he complained of pains all through his body. He was anæmic, his heart action feeble, but showed no evidence of cardiac disease. Examination of his lungs proved negative. His conversation was perfectly rational. About twenty-four hours after admission his condition entirely changed; he became delirious, walked in a tottering, feeble manner until he was put to bed. Temperature 101; pulse 62; respirations 30. The next day his temperature fell to 97.4; pulse 90; respirations 40. Examination of his urine was negative. No paralysis could be made out; his pupils were equal and responded both to light and accommodation. He soon sank into a condition of coma and died. His temperature again became elevated, reaching 105° before death.

Autopsy.—Some bands of adhesions were present near the base of the left lung, also the same on the right side.

The heart was hypertrophied, and in a slightly atheromatous condition at the valves, most marked at the aortic leaflets. The orifices were dilated, the tricuspid opening was markedly so, and fat clots were found in the right auricle. The stomach, intestines, and spleen were normal. The kidneys were large and congested; the capsules non-adherent. The dura mater was firm and slightly thickened. The pia mater was thickened and opaque and of a dirty yellowish color. The whole surface of both hemispheres was covered with organized lymph, also the base of the brain and cerebellum. The pia could be removed without much decortication. The lateral ventricles were slightly dilated.

Asylum Notes.

THE LUNACY COMMISSION'S REPORT OF THE STATE OF NEW YORK.

The first report of the new State Commission in Lunacy, which succeeded to the powers and duties of the State Board of Charities in respect to the insane, and of the former single Commissioner in Lunacy, which was recently presented to the Legislature, is a document of unusual interest and importance, since it deals with live questions of great moment to the people, and especially to the medical profession, of the State. Especially will its treatment of the subject of State care for the insane, to a discussion of which the major part of the report is devoted, attract wide attention and produce a lasting effect.

It classifies and enumerates the institutions wherein insane persons are kept; sets forth the scope of the Commission's work as prescribed in the act creating it (Chap. 283, Laws of 1889), and briefly, but with sufficient fullness, discusses these topics: Registration of the Insane, State Asylums, Revision of Statutes Relating to Insane, Official Responsibility, Private Patients, Discharge of Insane from Custody, Laws Relating to Chronic Insane, Transfer of the Insane, Removal of the Insane from their Homes to Asylums, Service of Legal Papers on the Insane, Discharge of Public Patients on Bonds, Habeas Corpus, Insane State Paupers. It then takes up the theme of State Supervision of the Insane, and recognizing its paramount place in the present bent of the public mind concerning insanity as a practical matter of State concern, it occupies some fifty pages with a full and explicit exposition of facts, views and opinions relating to the question at issue between the exclusive care by the State of all its insane on the one hand and what is called the "mixed system" of State and County care on the other. After adverting to the nature and gravity of the issue and the need of some fixed and permanent policy, the Commission gives some pages to considerations which it thinks properly precede any rational discussion of the matter. These are grouped under three main heads, viz.: 1st, the fact that insanity is a disease needing treatment; 2d, the error, almost universal half a

century ago and still common, that there is a hard and fast line of distinction between "acute" and "chronic" insanity justifying the belief that all or nearly all cases of the latter type are hopelessly incurable; and 3d, the wrong, unjust and injurious idea that those of the insane who are cared for at public expense are "paupers" in any true sense of the term, and ought not to be treated on any better footing than sane "paupers." It reviews the progress of efforts in this State to alleviate the condition of the insane, quoting from the act establishing the State Asylum at Utica in 1843, which forbade the counties (except New York, Kings and Monroe) from caring for their acute insane; and then, after showing the steps that led to its enactment, citing part of the Willard Asylum Act in 1865, which forbade the counties (with the same exceptions) from caring for their chronic insane. Their definite declaration of the State's purpose to care for all its insane has never been withdrawn, but, as a temporary expedient, forced upon the Legislature by the imperative necessities of the case. In 1871, power was given the State Board of Charities to exempt counties which in its judgment had sufficient facilities for caring for their own insane. This power has been exercised in behalf of nineteen counties, and one (Clinton) was exempted by special act. On October 1, 1889, there were 5,371 of these so-called "chronic" insane—3,138 in State asylums, 1,848 in exempt county poor-houses, and 385 in non-exempt county poor-houses. After discussing what should be regarded as essential requirements in any proper system of care for the insane, the following are presented as the leading points of advantage claimed for it by advocates of "county care:"

That the "chronic" insane being "paupers" and incurable, the lowest rate at which they can be comfortably fed, clothed, warmed, etc., is justifiable. 2. That they should be kept in the county of their residence, to have the benefit of visits from relatives and friends. 3. That they are better off in the county poor-houses, because land there is usually cheap and fertile, and they can be profitably employed in tilling it. 4. That better results may be looked for in small than in large institutions. The question whether these claims are sustained in actual results as observed by the Commission is treated exhaustively, and the verdict is emphatically in the negative. The several features of care that are nowadays regarded as essential in the proper treatment of insanity are reviewed, and in nearly every one it is declared that the county institutions fall lamentably below the reasonable standard that should be maintained. Some

striking, indeed shocking, instances are related of gross neglect and disregard of common decency and humanity : for instance, putting two filthy patients in one bed, bathing three or four patients in one water, sending out a debilitated man in chains to work in the fields, the care of insane women by male attendants, etc., and other like examples of flagrant impropriety, to speak mildly. Respecting the relative cost of the two systems, the actual difference is believed to be small ; while if equipment, facilities, attendants, medical supervision, etc., were required of county poor-houses in degree equal or approaching to those supplied at State asylums, the cost in the former would materially exceed that in the latter. The Commission recommend : 1st. That all the insane except in New York and Kings Counties be transferred to State asylums as soon as practicable. 2d. That districts based on proximity and population be assigned to each asylum. 3d. That inexpensive buildings be erected, at a cost not exceeding \$550 per patient, on the asylum grounds of the present State asylums. 4th. That the State pay all expense of maintenance and removal. 5th. That the Legislature appropriate money at its present session to begin the erection of such buildings.

The increase of insanity and necessity of additional provision for idiots of the unteachable class are briefly referred to, and various recommendations are made.

The number of insane in custody on the first day of October, 1889, was as follows :

State asylums, - - - - -	5,442
Counties of New York, Kings, and Monroe, - - -	6,970
Exempted counties, - - - - -	1,848
Non-exempted counties, - - - - -	385
City alms-houses, - - - - -	6
Quasi-public, - - - - -	541
Private asylums, - - - - -	315
<hr/>	
Total (an increase of 601 for the year), - - -	15,507
State Asylum for Idiots, - - - - -	477
Custodial Asylum for Feeble-Minded Women, - -	250
<hr/>	

The aggregate population of the foregoing is 15,507 insane, 477 idiots, 250 feeble-minded women.

The discussion of the question of State supervision of the insane concludes as follows :

"The conclusion of the Commission regarding the system of county care of the insane is, that however feasible in theory, in practical operation it has been found to have failed and fallen far short of the hope entertained for it when the act of 1871, sanctioning its trial, was passed. As a system it has developed inherent difficulties and defects which seem to be ineradicable, and which make its successful operation in all essential respects impossible. Such being the case, it ought to be abolished and the policy of State care for all of the insane, both chronic and acute, should be re-established at the earliest practicable date. It can not be said that the system of county care has not had a fair trial. It has been in vogue since 1871 under exceptionally advantageous circumstances. During all that time it has had the advantages of State supervision, and yet it has failed to meet every reasonable or just expectation. If the system has been a failure for nearly twenty years, is it not reasonable to conclude that it is likely to be a failure for all time to come. It is not claimed that the system of State care as now conducted is perfect, but it is steadily progressive; it is humanely and intelligently administered; it represents all that is best in the present state of medical knowledge; and whatever other criticism may be passed upon it, it certainly can not be said that the inmates of the State asylums are not comfortably housed, sufficiently clad, properly fed, provided with sufficient attendance and care, and given medical supervision and treatment of an exceptionally high order."

The report is signed by Carlos F. MacDonald, M.D., Chairman; Goodwin Brown and Henry A. Reeves, Commissioners.

A RESOLUTION RECENTLY PASSED BY THE NEW YORK STATE COMMISSIONERS OF LUNACY

"*Resolved*, That hereafter no license for the establishment and keeping of an asylum for the care, custody or treatment of the insane or persons of unsound mind, for compensation or hire, shall be granted except to a duly qualified medical practitioner of recognized professional skill and standing, who is a graduate of a legally incorporated medical college, and has had actual experience in the care and treatment of the insane."

New Instruments.

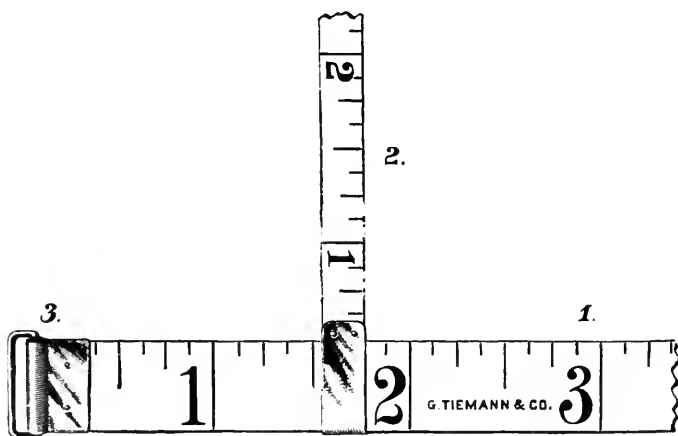
AN IMPROVED TAPE-MEASURE.

By WILLIAM C. KRAUSS, M.D.,

BUFFALO, N. Y.

Among neurologists perhaps no symptom is more important and significant than muscular atrophy or wasting. The keynote of diseases affecting the anterior columns of gray matter and of peripheral nerves, it is often improperly noted and erroneously measured.

The custom of comparing the two sides by sight or by touch, in many cases permits of errors which may be of serious moment in reaching a diagnosis or prognosis. The employment of the ordinary tape-measure, a step approaching accuracy, also permits of discrepancies. The practice of taking measurements at the lower, middle, and upper third of the extremities may be sufficiently exact for one measurement on one side, but when double measurements



are necessary for comparison, or when successive measurements are required, this mode is also inadequate, inasmuch as it is very improbable that the tape will be applied at exactly the same place as before.

To correct this difficulty, Messrs. Geo. Tiemann & Co., of New York, have made for me a tape-measure which permits of the greatest accuracy possible, and the absolute exclusion of guess-work in using it.

It is particularly adapted to the measurement of the extremities, and consists of a tape (1) thirty-six inches long and one-half inch wide. The English scale is graduated on one side and the metric scale on the other. The head is supplied with a swivel (3), through which passes the free end of the tape, permitting of uniform tension, greater accuracy in reading, and of its being held with one hand.

The second tape (2) is eighteen inches long and one-quarter inch wide, and is provided with a sliding head through which the first tape passes. This tape is therefore at right angles to, and movable upon, the first tape. It is also graduated after the English and metric scales. The object of this tape is to ascertain at what distance from a certain fixed bony point the first tape has been applied, so that on succeeding occasions the measurement may be taken at the same point. To illustrate: If the tape (1) be applied to the arm at a distance of five inches from the internal condyle of the humerus (reckoned by means of tape 2), it is obvious that, on succeeding occasions or in the comparison of the two extremities, the tape (1) must be applied at exactly the same point, thus excluding all possible chance of error.

I believe this tape possesses certain points of value to neurologists, surgeons, and those intent upon accuracy and precision in their observations.

176 FRANKLIN ST.

ELECTRICAL BATTERIES.

In the "Electrical World," Nov. 23, 1889, may be noted an account of the latest improved physicians' combination cabinet battery, of Jerome Kidder Manufacturing Co., of this city, a forty-point selector, by which any number of cells can be placed in circuit.

It contains also a faradic combination-coil (with slow and fast interrupter), resistance-coils, accurate milli-ampere meter, etc., making a most complete instrument for office use.

High commendation is also given to various other batteries, cautery, and surgical instruments.

It may be noted that, at the last electrical exhibit of the American Institute Fair, this firm were awarded the medal of superiority, which they have regularly received every year since 1872.

NOTICE.

INVITATION TO THE TENTH INTERNATIONAL MEDICAL CONGRESS.

In accordance with the decision of the Ninth Congress at Washington, the Tenth International Medical Congress will be held at Berlin from the 4th to the 9th of August, 1890.

By the delegates of the German Medical Faculties and the chief Medical Societies of the German Empire, the undersigned have been appointed members of the General Committee of Organization. A Special Committee of Organization has also been appointed for each of the different sections, to arrange the scientific problems to be discussed at the meetings of the respective sections. An International Medical and Scientific Exhibition will also be held by the Congress.

We have the honor to inform you of the above decisions, and at the same time cordially to invite your attendance at the Congress. We should esteem it a favor if you would kindly extend this invitation to your friends in medical circles, as way may offer.

DR. RUDOLF VIRCHOW,
President.

DR. VON BERGMANN,
DR. LEYDEN,
DR. WALDEYER,
Vice-Presidents.

DR. LASSAR,
Secretary-General.

All communications must be directed to the General Secretary, Berlin, N. W., Karlstrasse 19.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

NOTES ON SOME CASES OF CHOREA AND
TREMOR.¹

Case I.—Hereditary (Huntington's) Chorea in a Negro.
Case II.—Hereditary (Huntington's) Chorea, Apparently with Absence of History of Heredity. *Case III.—Hereditary Chorea with History of Chorea in Father and Infant Child.* *Case IV.—Paramyoclonic Chorea, with a History of Other Cases in the Same Family.* *Case V.—Tremor of Paralysis Agitans.* *Case VI.—Tremor of Disseminated Sclerosis.* *Case VII.—Tremor from the Abuse of Alcohol and Tobacco.* *Case VIII.—Diffuse Undulatory Tremor.* *Case IX.—Diffuse Undulatory Tremor.*

Service of DR. CHARLES K. MILLS at the Philadelphia Hospital.

Reported by J. L. BOWER, M.D. Resident Physician.

IN the Philadelphia Hospital Wards for Nervous Diseases are many cases illustrating diverse forms of chorea and tremor. Some of these have already been reported; others are examples of types of disease which have been so thoroughly studied and recorded that details of them would probably not serve a useful purpose; although a complete investigation of all, with sphymographic tracings, and close descriptions of the peculiarities of movement, might constitute a valuable contribution.

¹ Presented to the Philadelphia Neurological Society, January 27, 1890.

Condensed notes will be given of a few cases with brief comments, particularly directed to the observed peculiarities of tremulous or choreiform movements. Of the nine cases here reported five have been observed in the Nervous Wards of the Philadelphia Hospital ; the other four have been furnished me by Dr. Mills, from notes of cases observed elsewhere. One of the most interesting of the cases (Case IV.) has been in the Hospital for a considerable time and has been frequently studied and commented upon by the physicians in attendance. Dr. James Hendrie Lloyd gave a sketch of this case in a clinical lecture recorded in the *Medical and Surgical Reporter*, May 19, 1888 ; but it is sufficiently important to call for a more detailed report. Cases X. and XI. would seem to be practically undescribed forms of fibrillary tremor, as exactly similar cases are not to be found in the text-books. Dr. Mills has suggested to designate this form of tremulous movement *diffuse undulatory tremor*. The cases of paralysis agitans, disseminated sclerosis, and alcoholic tremor are introduced to compare the varieties of tremor.

CASE I.—Hereditary (Huntington's) Chorea in a Negro.

E. H., age 36, barber. His father died of some disease unknown to the patient. The disease from which he is suffering is inherited from his mother ; she with two sisters and one brother were or are suffering from the same trouble. In his mother the disease developed at the age of 26 and gradually grew worse until she died at the age of 68 from exhaustion ; he does not know exactly at what age it developed in his aunts and uncle, but thinks that it was somewhere between 20 and 35 ; one of the aunts is still living at the age of 50 years, the other is dead ; the uncle is 42 years of age and in him the arms alone are affected.

The patient has been a hard drinker ; when 28 years old he was drinking heavily and became suddenly paralyzed in both lower extremities and remained in this condition for three months, when power gradually returned. From description the affection does not seem to have been multiple neuritis, as the paralysis came on suddenly and he had no pain. Three years ago he had a venereal sore, probably a chancre, followed by a bubo.

Six years ago he first began to have twitching of the hands ; this spread gradually but very slowly, until his

whole body became affected and his present condition reached. When he stands he sways a little from side to side, occasionally lifts one or the other foot, raises and depresses his shoulders, almost constantly opening and shutting and contorting his fingers, at times also thrusting one or both hands to his back. He is never entirely still, and he gives the impression that he is trying hard but unsuccessfully to control his nervousness. When he walks his gait is stiff, weak and unwieldy (perhaps in part from the old paralysis). The irregular movements in the hands and fingers continue, and in the extremities he has occasional swaying and pseudo-ataxic movements; now and then he lifts one foot high and balances himself before stepping out. When sitting down he is still unquiet, but in less degree; his trunk, arms and hands are moving almost continuously, the latter being shifted from his knees to his thighs where he places them in order to keep them quiet. All his movements consist of a series of irregular starts affecting one part after another or several parts together. At times they have the appearance of design, and the patient seems also to be trying to restrain them. The movements are not increased by voluntary efforts, but seem to be by excitement. He writes fairly well, but slowly and with difficulty, and it appears to be a great effort to keep himself in the proper position and direct his movements for writing. His signature is given below.

A handwritten signature in cursive script, reading "Edmund Harrison". The letters are fluidly connected, with a prominent loop at the end of the last name.

His speech is slow, and at times he pauses as if to co-ordinate the movements of articulation.

On inquiry it is found that the parents and ancestors of the patients were all full blooded negroes. The patient's mental condition so far as can be determined, is not impaired. His physique is good; height, five feet nine inches; weight, 130 pounds. His lungs, heart and kidneys are normal.

This case is interesting for several reasons. In the first place, it is another illustration of hereditary chorea. Its occurrence in a negro adds to the interest, as chorea of any type is rare in this race. His case presents points of great similarity to that of Miller, a patient in the same wards, an account of whose disease and remarkable family history

has been given by Dr. Wharton Sinkler, in one of the best papers on hereditary chorea.¹

The next case, so far as the choreiform symptoms are concerned, would seem to be best classed as Huntington's chorea, but the most careful inquiries, could elicit no history of heredity. The patient's mental condition is somewhat impaired, but not to a marked extent, and he gives a clear history of the absence of nervous disease in his family. He would be able to recall the striking fact of a disease similar to his own having occurred in his family. His parents, brothers and sisters, were certainly not so affected.

CASE II.—Hereditary (Huntington's) Chorea, apparently with absence of history of heredity.

H. C., age 32, a painter, to the coming on of his present disease had excellent health. He is a moderate drinker, and denies venereal trouble; he does not use tobacco. His family history is entirely negative.

About three years ago he began to have pain in the right knee which was swollen; in about a month he recovered from this trouble, but shortly afterwards he noticed that his hands and his fingers were weak, and sometimes trembled or twitched; his legs and head also became affected about three months after his upper extremities.

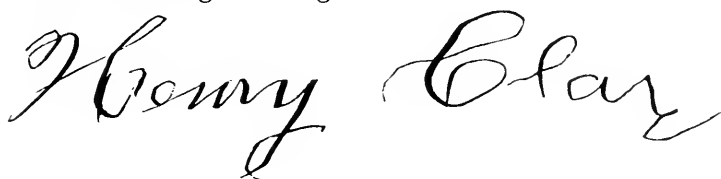
When sitting down his legs and feet are usually quiet, but he has a most constant though not very marked movement of his head, trunk, and upper extremities; his head is moved a little backwards or from side to side in an almost rhythmical manner; his trunk is occasionally lifted or twisted as if about to shrug his shoulders; he carries his left arm across his chest, the hand and fingers partially extended; his right arm generally hangs by his side at full length; both arms are kept comparatively quiet, but occasionally are shifted about uneasily; his fingers and hands are constantly moving, going through a series of irregular or athetoid movements—perhaps one or two fingers are flexed while others are extended, or one or all fingers may be extended partially or fully; sometimes the fingers are thrust apart or sometimes the thumb and fore-finger are brought together and then separated. If the hands are supported on the knees, the movements lessen considerably; occasionally he shifts the position of his hands or moves his fingers up and down.

¹ Journal of Nervous and Mental Disease, February, 1889.

When he walks his choreiform movements are much increased. Athetoid movements of the fingers are especially marked. He walks with a sort of twisting or half rotating movement of the trunk and limbs, occasionally lifting one foot high in the air and bringing it down with a flourish, and every few steps pausing as if to poise himself. By a strong effort of the will the movements seem to be lessened. Voluntary effort such as writing, or conveying a glass of water to his lips, cause the movements to be less marked. Excitement of any kind greatly exaggerates his movements. He cannot protrude his tongue fully, and loss of power or control of the tongue seems to be of the same character as that shown in other parts of the body.

Knee jerk and muscle jerk are considerably exaggerated; slight ankle clonus is present on both sides. He has no loss of sensation. During sleep the movements cease entirely. His hand-writing is quite passable, but when he writes he seems to be putting forth extraordinary effort.

The following is his signature:

The image shows a handwritten signature in cursive script. The name 'Henry Clay' is written in a fluid, elegant style. The 'H' is large and loops around the 'e'. The 'C' in 'Clay' is also large and loops around the 'l'. The signature is written in dark ink on a light background.

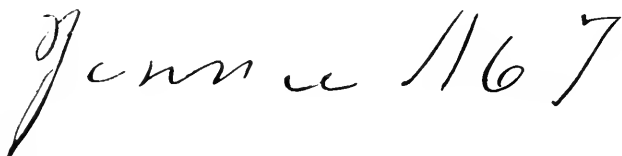
His speech is slow but quite intelligible; it seems to require an extreme effort to co-ordinate the movements of articulation. He can read fairly well by holding the book against his chest to steady it. His general health is excellent; height 5 feet 11 inches; weight 145 pounds; lungs, heart and kidneys are normal.

CASE III.—Hereditary chorea, with history of chorea in father and infant child.

The notes of this case are meagre, but it is worth while to report it as it adds another authentic record to the statistics which demonstrate the peculiar hereditary character of this form of chorea. The patient, J. M., married, white, was 41 years old at the time of coming under observation. Slight twitchings or choreiform movements began five years before, that is, when she was 36 years old. The disorder gradually increased. The movements were decidedly like those shown by the other cases of hereditary chorea reported in this communication. They affected the head, arms, legs, and speech, much more markedly on the right than on the left side. The patient ate well, her sleep was broken, and her mental condition was one of

slight beginning dementia. Her father was affected with the same or a similar disease. He was a hard drinking man. She had a baby only four months old, almost constantly affected with choreic movements of both hands.

Signature:



The following seem to be the peculiarities of movement and tremor in these cases of so-called hereditary chorea: The movements are usually first noticed in the upper extremities, especially in the hands. The disorder spreads slowly and gradually, but very surely, to other parts, until the entire body is more or less affected, the upper extremities commonly continuing to exhibit the symptoms in the most marked degree. Excitement increases, voluntary effort decreases the movements. When sitting or lying down the patient is much quieter than when standing or walking. Speech and writing are both affected, and as the disease advances require great effort on the part of the patient. Only in its very early stages would this disease be likely to be confounded with any of the other forms of tremor or chorea. Very early it is conceivable that even disseminated sclerosis might be confounded with it; but early or late the tremor of disseminated sclerosis is usually more confined to particular parts; and in disseminated sclerosis voluntary effort markedly increases the tremor, while it has the opposite effect in hereditary chorea. Emotion aggravates both disorders. When the head is affected in disseminated sclerosis the movement is a more or less uniform oscillation, while in hereditary chorea the head is moved about in an irregular manner—forwards, backwards, or to one side, as if the exertion was partly under the control of the patient's will.

CASE IV.—Paramyoclonic chorea, with a history of other cases in the same family.

C. E., aged 21, huckster, had three sisters who died of phthisis, at the ages of 4, 18 and 19 respectively. One brother died two years ago and was affected with the same disease as the patient, the age at which it developed and the causes not known. He has two brothers living, and he says that one of them has the same disease, only not so far advanced. In this brother the affection began at the age of 20 with no apparent cause, and has now existed for

over a year. His other brother is perfectly well. No history of nervous disease on either paternal or maternal side could be elicited. The patient says that before the advent of his present disease he was always healthy, never used tobacco, or intoxicating drinks, and had no venereal history.

Three years ago while huckstering his horse became frightened and ran away. When he found that the horse was beyond control he jumped from the wagon, hurt himself, and became unconscious; on coming to his senses his whole body jerked as it does at present although not so markedly. He was carried home and put to bed and remained there for five weeks, at the end of which time he was able to walk with the aid of canes, but frequently fell down. At present he is unable to walk and must be carried from the bed to his chair, in which he must be tied or he would be thrown to the floor by the excessive, inco-ordinate, involuntary, universal movements. He is unable to feed himself because of jerking of the arms. His appetite and general health are excellent; he sleeps well and during sleep is frequently quiet. It is impossible to obtain knee jerk as the limb becomes rigid when an attempt is made to elicit it. He has slight left ankle clonus. He has control of bowels and bladder. He shows no impairment of sensation or of the special senses.

The following is an attempt at a description of the character of the choreiform movements: When lying quiet, not watched or spoken to, or excited in any way, he is comparatively easy and may be perfectly quiet for several minutes or even much longer. Occasionally he will raise one or both hands slightly or it may be place them on his breast, or he may flex and extend the fingers in an irregular manner. These movements are made slowly and at intervals, but usually from six to twelve in a minute. Excitement or emotion, or attempted voluntary effort, greatly increase the movements and they rapidly become general; they are violent, irregular, inco-ordinate, wild, and shock-like; they are more or less independent of each other, numbering from sixty to eighty per minute. During the excitation the body is thrown forward and backward, the head is moved from side to side and often thrown forward and backward quite strongly. The facial muscles are usually free except the orbicular muscles of the eyelids. His upper extremities move rapidly, and usually irregularly and independently of each other. With the irregular movements he has at times certain automatically repeated acts. The most frequent of these seems to be a brushing of the head and face with the hands either open or closed, or a slapping

of one hand in the other, or on the knee. Frequently he throws his arms out at full length, then rapidly across his chest and face; or he may clasp his hands for a moment or execute some other irregular movement in the lower extremities, not so marked as in the upper, but otherwise very similar. He flexes and extends his thighs, legs, and feet in the most irregular manner; occasionally his legs are extended perfectly straight for a few seconds in what appears to be a tonic contraction, but it is due to a very rapid clonus. When he is lifted from his bed his body becomes rigid as a board; his arms are thrown wildly about, and his lower extremities become almost immobile by the extreme rapidity of the muscular contractions of the body and lower extremities. When he is placed on his chair the violent contractions jump him up and down for a minute or two. His speech is interfered with apparently through difficulty in co-ordinating the muscles of articulation, but when he commences to speak he talks quite rapidly.

The mental condition of the patient seems to be unaffected by the disease, and his general physical condition is good.

A careful consideration of this case shows points of great similarity between it and cases of hereditary chorea, although at first sight this resemblance might not attract attention. The movements are similar although far more violent and general. The history shows other cases in the same family. The case differs from hereditary chorea in having begun acutely, and at an earlier age than is common in the latter disease; also in the much greater rapidity and violence of the inco-ordinate movements.

CASE V.—Tremor of Paralysis Agitans.

A. K., age 54, a weaver, for sixteen years has been troubled more or less with chronic rheumatism, principally in the shoulder-joints. He is a very moderate drinker and smoker, and has no history of venereal troubles.

About five years ago he first noticed a weakness in the right arm, and this has steadily progressed. Three years ago a tremor of the right hand and fingers first became manifest; at first it was not constant and was more marked when under excitement. About one year ago the right foot "trembled" also, and within a month the left hand developed a fine tremor which is not always present. This tremor is fine and rhythmical, and never ceases except during sleep. By an effort of the will it can be lessened but not stopped; voluntary effort as in drinking from a glass, very much lessens it; excitement increases it con-

siderably. Grasping the arm lessens it decidedly but does not cause it to cease. If the hand is rested on the knees the tremor continues but is less marked. There is slight stiffening of the right hand, and he writes slowly and with considerable effort, but comparatively well.

The patient's shoulders are stooped, the head carried toward the chest and held fixed as if in a stock. The face is fixed, dull, and expressionless. He shows nothing peculiar in his gait. Sensation is not impaired.

Signature :



Several cases of paralysis agitans, more typical, or at least more advanced, than the one here reported are in the Nervous Wards of the hospital, but this case was selected for report and comparison because at a stage, so far as the tremor is concerned, when, if ever, it might be confused with some other affection.

In the main, this case affords the diagnostic features usually given by the books; the tremor is lessened by an effort of the will; it is, however, increased by excitement. It is fine and rhythmical, and fixity of limb and feature are present.

CASE VI.—Tremor of Disseminated Sclerosis.

J. O., age 71, laborer, ten years ago had mild malarial and four years later break bone fever, both of these attacks persisting for about five months. Up to ten years ago he was a hard drinker. He is a heavy user of tobacco, both chewing and smoking. He has no history of venereal troubles.

Two years ago he noticed a tremor of the right hand which first became apparent when he attempted to use the hand as in eating; this gradually became so marked that when possible he rested his right hand and used the left one. In about two months he noticed that the left hand also trembled, and shortly after he felt the same tremor in his body and lower extremities, although not so marked.

When the patient sits with his hands on his knees they are comparatively steady, but occasionally a fine tremor is seen. When his hands are extended without support, the

tremor is quite marked in both but more apparent in the right hand. When he attempts to control his movements by mental effort, the tremor is considerably exaggerated. If the hands or arms are grasped the tremor continues, and can be felt involving the entire limb. A marked increase occurs if a voluntary effort is made, as when he attempts to drink from a glass—the tremor then becomes wide, coarse, irregular and jerky, and in efforts to carry the glass to his mouth much water is spilled. There is a fine oscillation of the head, chiefly in a lateral direction, and occasionally a slight spasm or twitching of the zygomatic muscles of the right side of the face. The tremor involves the trunk slightly; he has also tremor of the tongue.

Nothing particular is noticed about his gait except that it resembles that of paralysis agitans in that his head is slightly bowed; his face has rather a blank and listless expression, but this may be accounted for by his age and low intelligence; he complains of weakness of both upper and lower extremities, especially of the right arm. The dynamometer shows a grasp of 26 for the right, and of 56 for the left hand. The least exertion fatigues him. This weakness first became manifest seven months ago and is steadily growing worse. There appears to be no impairment of sensation; the pupils are equal, moderately dilated, and respond to light; he has no nystagmus. Knee jerk is abolished and slight ankle clonus is present on both sides. He is frequently troubled with headache and vertigo, the latter being most marked in the morning. He speaks in a slow and tremulous manner. He has perfect control of the bladder and rectum.

CASE VII.—Tremor from abuse of tobacco and alcohol.

J. R., aged 63, about a year ago began to be afflicted with tremor in both hands, which has become gradually worse during the last three months. He complains of feeling dizzy, and if he turns suddenly is in danger of falling. He has no headache, nor any pain in the body. He sleeps well but has no appetite. For the past thirty years he has been accustomed to drink several glasses of beer and smoke twelve pipes during a day.

The tremor affects both hands about equally, it is coarse but not large, and keeps up continually without any variation; it is sometimes increased by exertion. The arms as well as the hands are somewhat affected. Sometimes the tremor almost entirely disappears for one or two days; and in the latter part of the day is generally better than in the morning.

Under enforced abstinence from alcoholic drinks and

tobacco, in about two months this man's tremor almost absolutely disappeared. The peculiarities of the tremor were its bilaterality, its complete discontinuance at intervals, its greater regularity than in disseminated sclerosis, its better response to treatment, and the absence in the patient of the other symptoms and conditions which are usually associated with paralysis agitans or disseminated sclerosis.

CASE VIII.—Diffuse undulatory tremor in a case of chronic spinal degeneration.

C. J. H., age 53, had had some pain in the lower part of the back for years, after which he noticed trouble with his feet when walking down hill. He grew gradually worse, but had no history of true pain, only cramp or spasm in the calves.

He cannot lift his toes in walking, stumbles and feels his feet under him. He has no trouble with his bladder or bowels. Twelve years ago he had some confusion of sight, but was relieved by glasses; he has no diplopia. The knee jerks are exaggerated. He has advanced paresis of both legs. The back muscles are not rigid. In the arms he has no palsy but a jerking or twitching of the muscles. For four months he has had cramps in the legs, and in the arms, but not so marked; also a dragging sensation on the left side of the lower part of the abdomen. He has, at times, a tingling sensation all over his legs, and sometimes in his arms. He has "sore" feelings in the thighs and legs.

A marked and very peculiar feature in this case is an almost universal tremor of the muscles of the thighs, legs, arms, forearms and trunk. Testing him with electricity it was thought at first that the tremors were contractions produced by the electrical application. Wave-like movements are present all the time. The surface of the body presents the appearance of a gently undulating sheet of water. In the shoulders, arms, etc., almost every physiological movement of the muscular groups could be seen; the muscles could be picked out by these involuntary contractions.

CASE IX.—Diffuse undulatory tremor in a case of chronic spinal degeneration.

G. B., 31 years old, white, married, two years before coming under observation noticed that his left leg began to fail, and six months later his right became similarly affected. In a few months the loss of power in the left lower extremity was very extensive; at the time of examination all the foot movements being abolished and those of the leg and thigh much weakened. The right leg and the left upper extrem-

ity paretic, but still retain considerable power. Knee jerk on each side are retained, muscle jerk increased; farado-contractility is retained, but somewhat diminished in some of the atrophied muscles of the left leg. Sexual power is good; he has no affection of the bladder or bowels and no anæsthesia.

The marked peculiarity of this as of the last case is the presence of a diffuse undulatory tremor, affecting both legs very generally.

The tremor in these cases is similar to that observed in the tongue in some bulbar strophic cases, and doubtless its pathology is the same as in these, but it is very rare to observe this extreme and widespread undulatory muscular tremor affecting parts other than the tongue.

EXOPHTHALMIC GOITRE.

Before a branch of the British Medical Association, reported in the *British Medical Journal*, Jan. 4, 1890, Dr. Cheadle showed a case of exophthalmic goitre in a male subject, in whom recovery was maintained for more than twenty years. He considered the symptoms due to disorder of the medulla, of the upper cervical portion of the cord, the region of the cardio-inhibitory, accelerator, vasomotor, vomiting, and glycogenic centres, all of which were—some constantly, some occasionally—involved in the disease. In February, 1868, the patient suffered from characteristic vascular excitement, marked exophthalmia, enlarged thyroid, which pressed so severely on the trachea that the consequent dyspnoea compelled the patient to sleep sitting in a chair. Tincture of iodine was freely given, followed by improvement in the course of a week, and this had continued and steady. The tendency of the disease seemed to be toward slow recovery in from one to five years. Cases were on record from death from pressure on the trachea, from acute mania, from cerebral hæmorrhage, from pulmonary congestion and anasarca, and from various intercurrent affections. One of the greatest dangers Dr. Cheadle considered to be persistent vomiting with diarrhoea. Absolute rest was essential in the acute stage, and opium with digitalis or belladonna. Galvanism was useful in the chronic state, but caused undue excitement in the acute form.

L. F. B.

CONGENITAL BILATERAL PLEUROPLEGIA AND FACIAL PARALYSIS.¹

By A. SCHAPRINGER, M.D.,

NEW YORK.

BECKY F., æt. eight years, was brought to the Eastern Dispensary of this city by her fourteen-year-old sister, on October 11, 1889, because of a cough that was accompanied by a muco-purulent expectorate, and was particularly troublesome at night. She was directed to the department for diseases of children, but by mistake went to my department, that for diseases of the eyes. Thus it happened that she came first under my observation. The



peculiar appearance of her face impressed me at once and lead me to make a careful examination, the results of which are detailed below.

Dr. H. Koplik, the attending physician to the department for the diseases of children at the dispensary, at my request, kindly turned the case over to me, and for this great favor I here again take occasion to thank him.

¹ Case presented at the meeting of the New York Neurological Society, December 3, 1889.

The disease of the respiratory tract for which the little patient sought medical aid stood in such distant relationship to the remarkable conditions which are described below, that I shall pass it over without any further reference.

The child is of a development corresponding to her age. The deposit of subcutaneous adipose tissue is rather scanty. She is possessed of the average intelligence. The circumstances of the family have been such as not to have permitted them to send her to school, and as a consequence she has not yet learned to read. When walking, she stumbles and falls more frequently than other children of the same age. She declares positively that this is because of a weakness in her legs and not on account of a lack of appreciation of the irregularities and obstacles on the surface of the floor or sidewalk.

She generally holds her head rigidly erect. The glabella is bulging, but otherwise there is nothing unusual about the skull. Her face is pale, expressionless, and presents a mask-like appearance. The naso-labial fold is absent on both sides. There is not the slightest indication of folds in the integument of the face either in laughing or in crying. On one occasion, when she was giving utterance to some peculiar inarticulate sounds, I inquired why she was crying, and was immediately enlightened as to the nature of the sounds by her elder sister, she informing me that the little one was laughing and not crying as I had supposed. The patient cannot close her lips, and in speaking, substitutes linguals for labials, saying, for example, "tata" for "papa," and "nana" for "mamma." The right angle of her mouth is drawn downwards and outwards, and is the only place where any mimetical movements are noticeable. The little expression there is is to be perceived principally when she is talking. Whistling, blowing, etc., are absolutely impossible.

She cannot wrinkle her forehead, either vertically or transversely. When her eyes are open the ordinary and normal amount of eyeball is uncovered. Usually both eyes are open to an equal extent, but quite often one lid or the

other hangs a little lower than its fellow. When required, the eyes can be fully and completely opened, showing that the levatores palpebrarum superiorum are not involved. Complete closure of the eyes is impossible, although upon a forcible effort the lids may be so nearly approximated that only a very inconsiderable interval is left between them. To this condition of the lids may be attributed the fact that the eyes have been protected from irritation. There is on either side, quite prominent, that abnormal crescentic fold of skin, covering the inner angle of the eye, and, at v Ammon's suggestion, called the epicanthus. The caruncle and plica semilunaris are only slightly developed on both sides. They cannot be seen on a front view, and only become visible when looking from the side of the face towards the inner angle.

Ordinarily the axes of the eyes are parallel. Frequently, however, the right eye is directed a little more upwards and slightly outwards. If an object be held in the median plane a few feet in front of the child's head and then she be asked to fix her eyes upon it, she does so without difficulty. She is also able to follow the object with both eyes if it be lowered or elevated in the median line. If the object be approached to her face, keeping it in the sagittal plane, she follows it readily; and she will keep her eyes fixed upon a finger held near the nose for a longer period without fatigue than is generally borne normally. These, however, are all the movements of which the eyeball is capable. They are rotation upwards, downwards, and convergence. When an object is moved to either side of the median line and the child is told to look at it, she invariably answers, "I see it;" but she stares directly ahead, turning her eyes neither towards the right nor the left. This same condition obtains if the eyes be alternately covered by the hand. For example, if the right eye be covered and the object then moved out of the median line to the left, the uncovered eye does not follow the object, showing a paralysis of the external rectus of the left side. On the other hand, if the object be moved to the right, the eye does not follow it, showing a paralysis of the internal rectus of the same side.

As we have seen, the two internal recti contract freely if required, in order to bring the eyes into convergence for near objects. If, however, one of these muscles be required to act in unison with the external rectus of the opposite eye, for a conjugate lateral version there will be found an utter inability to do so. The two external recti muscles are either completely paralyzed or entirely absent, as neither the right nor the left eye can be directed externally from the median line the least particle. On the other hand, the internal recti are only relatively paralyzed. They converge the eyes readily when required, but they are utterly unable to respond to an impulse which under normal condition would induce a conjugate lateral version. When the child wishes to see an object situated to the side of the median plane, instead of rotating her eyes she turns her entire head.

Under normal conditions the range of binocular fixation (not to be confounded with the field of vision) may be said to correspond to a geometrical solid with three dimensions. In the case under consideration, however, the range of binocular fixation is reduced to a sagittally placed plane, and as such it has but two dimensions, one of which is vertical and the other sagittal.

With the ophthalmoscope the vessels in the fundus appear to be more tortuous than usual. Otherwise nothing worthy of mention is to be seen.

The left eye is practically emmetropic, having only a slight degree of astigmatism. The right eye is myopic to the extent of about 3 D. This is the eye which, as was stated before, is directed upwards and slightly outwards when looking at a distance. In order to avoid losing the good will of the child I refrained from carrying the rather wearying tests for the determination of the acuity of vision out to completeness. As far as could be determined, V. was nearly, if not quite, normal in both eyes.

The ophthalmoscopical observations were conducted without the aid of mydiatics, and the little patient's efforts of accommodation proved a great obstacle. They showed, however, that the functions of the ciliary muscles were

unaffected, which could also be demonstrated in other ways.

With reference to certain analogous cases reported by other observers, it would be well to state explicitly that there is no strabismus convergens, and that there is not the least trace or scar of a former tenotomy or myotomy, and that, furthermore, in looking downwards there is no abnormal convergence of the axes of the eyes.

The size and mobility of the pupils are perfectly normal.

When the tongue is protruded it is deflected a little to the left of the median line. The left half of the tongue is a trifle smaller than the right half.

Movements may be made in all directions, but not with the requisite completeness and force. When eating, the child is obliged to use her finger to dislodge food from her cheeks. She says that she is unable to masticate hard substances, such as bread-crusts. If a hard body is placed between her teeth, she is able to hold it there quite firmly. She can move the lower jaw laterally towards the right side, but not towards the left, indicating a paralysis of the right external pterygoid muscle. She has uvula bifida. The child herself called my attention to a remarkable anomaly in her mouth, which I should certainly not have thought of looking for otherwise. The cord-like structure, which may be felt by passing the finger along the buccal mucous membrane between the alveolar process of the upper jaw and the cheek in the neighborhood of the canine fossa, and spoken of by the dentists as the lateral *frenulum* or canine ligament, is enormously developed in this case on the right side, while on the left side the state of its development is normal. What significance can be attached to this condition I am not prepared to say, my opportunities for making anatomical investigations in regard to it having been too meagre. Hearing, taste and smell are normal, as is also the sensibility of the skin.

I wish to express my great obligations to Dr. Geo. W. Jacoby for helping me during the examination of the nervous symptoms of this case.

The distal phalanx of the left index finger is bent out of

the long axis of the finger at an angle of about 150° in the direction of the middle finger. The affected interphalangeal joint possesses the normal mobility, and there is not the least trace of a previous inflammation. All the circumstances point towards the defect being a congenital one.

The anterior aspect of the thorax shows an extraordinary deformity. The sternum is deeply sunken in, and presents a long vertical groove situated between two prominences formed by the bulging of the anterior portions of the sites on either side. Dr. Abraham Jacobi, who examined this deformity at my request, pronounced it a not very well-marked example of that form of imperfect development known in the literature of the subject as "funnel chest."

No other deformities, such as webbed fingers and toes are discoverable.

The parents of our patient are natives of Russia, where the patient was also born. They are divorced and live in distant parts, while the child and her elder sister are being brought up by an uncle. The parents are not consanguineous. The sister of whom mention was made before is physically well developed and looks healthy. Another child, however, of the same parents died at the age of a few months. The child who is the subject of this paper is said by her relatives to have been free from any anomalies at birth, except the before-mentioned angular index finger. The relatives also state that the distortion of the mouth was developed subsequent to an attack of convulsions which occurred in her early infancy. According to another version, the blame attaches to injury received by striking her head in a fall. It is perfectly manifest that no weight can be given to those accounts, and that we must consider the anomalies as congenital.

The most interesting features of the case are unquestionably the lesions of the nervous system. They include the motor branch of the trigeminus, the hypoglossus and the facial, but particularly those nerve tracts of both eyeballs that control the conjugate lateral version. I should like to confine myself to the consideration of this last anomaly entirely.

Previous to this there has been no simple and concise term to designate the condition of paralysis of the conjugate lateral version of both eyes to the right or to the left, or, as in this case, to the right and the left with preservation of convergence. I therefore suggest the name "Pleuroplegia," deriving it from the Greek word *pleuron*, the original meaning of which is "the side." The word is constructed similarly to "Pleurothotonus," by which we designate a convulsion that draws the body to one side and is seen in tetanus. In the succeeding portions of this article the reasons for the introduction of this kind of term will be shown to be timely as well as conducive to a clearer conception of the subject.

Pleuroplegia (or to be more exact, ophthalmopleuroplegia,) is generally seen in diseases of the pons, such as neoplasms, hæmorrhages, the softening due to arterial thrombosis, etc., and is usually unilateral. In attempting to explain this phenomenon we have to reason about as follows:

Given a case where there is left-sided pleuroplegia, with the power of convergence retained, the following parts must be undisturbed in regard to their anatomical structure and their functions: 1st. The two internal recti muscles. 2d. That branch of the motor-oculi that supplies these muscles. 3d. The two nuclei for the internal recti. 4th. The two intracerebral paths which connect the nuclei of the internal recti with the centre of voluntary convergence. This is supposed to be a single centre situated in the cortex. 5th and last. This convergence centre itself. In our supposed case the lateral conjugate movements toward the left side are completely arrested. This movement is accomplished by the external rectus of the left eye and the internal rectus of the right eye.

We shall now study the innervation of these individual muscles separately.

Since the left external rectus is completely paralyzed, the following series of neuro-muscular structures may be regarded as having been rendered inactive: 1st, the left rectus externus; 2d, its supplying abducent nerve; 3d, the

abducent nucleus in the pons; 4th, the nerve tract which connects this nucleus with that portion of the cortex of the right side of the brain in which the centre that presides over the voluntary left lateral version of both eyes is situated; 5th, this centre itself.

The right internal rectus muscle is only relatively paralyzed, that is only in regard to conjugate lateral version. There is no impairment of its power of convergence. If we now follow the chain that connects the right internal rectus with the voluntary centre of conjugate left lateral motion, we have: 1st, the muscle under consideration; 2d, its innervating branch from the right motor-oculi; 3d, the right internal nucleus. At the internal nucleus the impulses of convergence and of conjugate lateral version separate. For the internal nucleus has two lines of communication with the cerebrum. The one connecting it with the centre of voluntary convergence has been discussed, the other which will now engage our attention is that which connects it with the centre of voluntary conjugate lateral version. This centre is located in the cortex of the right hemisphere, that is on the same side as that internal nucleus to which we had come in the course of our progress from without inwards. The connecting path between the right internal nucleus and the centre of voluntary conjugate lateral version in the cortex of the right hemisphere is not, however, a perfectly straight one confining itself within the boundaries of the right cerebral hemisphere, but takes part of its course through the substance of the left hemisphere, making a sort of loop and passing quite close to the nucleus of the left abducens. This is the same abducens nucleus that we met before in the course of our analysis. So near to this nucleus do the connecting fibres pass that a lesion of the nucleus almost invariably involves them. The course of the fibres carrying the impulses of conjugate left lateral motion are undivided from their origin in the cortex of right hemisphere until they reach the neighborhood of the left abducens nucleus. Here, however, they split into two divisions, one of which joins the left abducen nerve, while the other passes over to the opposite side into the right

internal nucleus and by means of this communicates with its respective branch of the oculo-motor nerve. If the central communicating fibres are injured in any part of their course, there will result as a consequence an absolute paralysis of the left external rectus and a relative paralysis of the right internal rectus. In other words, a left-sided pleuroplegia. This would, of course, also follow from lesions involving either of the two terminals, that situated in the right cortex, and the left abducens nucleus.

What has been said in reference to left-sided pleuroplegia holds good *mutatis mutandis* of that of the right side.

That the term "paralysis of the abducens" for this condition is insufficient and consequently conveys an erroneous impression, is manifest, but still it is used in the literature of the subject. Perhaps the term "nuclear abducens paralysis" would be more correct. Even this, however, will not answer as a designation for the clinical symptom as such, because it makes a topical diagnosis, which, while it may apply to a large number of the cases, will not apply to all of them.

The terms "deviation conjuguee" and "paralyse conjuguee" remind us that we are indebted to a group of French investigators for the original clinical observations concerning this symptom. I shall only mention the discoveries of Achille Foville and Féréol. Foville first published, in 1858, the observation that in unilateral disease of the pons there is a paralysis of associated lateral motion of the eyeball toward the side on which the lesion of the pons was situated.

In 1872, Féréol called attention to the seeming paradoxical circumstance that the internal rectus responded freely to the impulses of convergence while it was powerless to carry on the motions of conjugate lateral version. The terms "conjugate deviation" and "conjugate paralysis" are both applicable to disturbances of motion in every possible direction, and it is always necessary to denote the direction, whether upwards or downwards or to the left, etc., by the appropriate words. Of all the conjugate paral-

yses that which we have called "pleuroplegia" is of the most frequent occurrence, and, because of the twofold rôle played by the internal rectus muscle, the most interesting.

In not every case where pleuroplegia or pleuroparesis exists is the condition to be discovered by any marked deviation of the eyes. This is well illustrated by the following case which came under my own observation :

A four-year-old girl fell down stairs, and was brought to the Eastern Dispensary. Dr. W. W. Van Arsdale, to whom she was first presented, asked me to examine her left ear from which there had been some bleeding. At the time we saw her the child was perfectly conscious and in good spirits. There was no eye symptom complained of, and nothing remarkable was to be seen. It chanced that I had had a case, a short time previously, of paralysis of certain muscles of the eye, and that led me to investigate the mobility of the eyeball in this case. The child when requested to follow a finger with her eyes, could only do so in the left half of the field of fixation. To the right she could only follow a trifle beyond the middle line, and there the eyes remained in a sort of nystagmus-like motion. There was a difference in the two eyes, inasmuch as the left eye went a little further beyond the middle line than the right. I know nothing of the future of the case, as the parents never brought her again.

For the sake of completeness it may be mentioned that the opposite condition to pleuroplegia, namely, a paralysis of convergence, with retained conjugate lateral motion, has been observed and is being regarded with increased attention in the newer literature. If a short expressive term for this symptom is desired, that of "mesoplegia" is suggested.

In the case described here the symptoms are evidently due to a lack of development involving the nuclei of the abducens, facial, hypoglossal and trigeminus nerves.

It is because of the close neighborhood of these nuclei that this seems the most reasonable explanation of the symptoms. The inconstant strabismus sursum vergens, alternately now in one eye and then in the other, denotes that along with the formative defect there is an active

unstable cause at work, in regard to whose nature I may be excused from theorizing.

Only four parallel cases have as yet been recorded in literature. They are the cases of "congenital bilateral abducens facial paralysis" of Alfred Graefe (Halle),² G. C. Harlan (Philadelphia),³ Julian J. Chisolm (Baltimore),⁴ and P. J. Möbins (Leipzig).⁵ The present one is the fifth case.

Of the five cases under consideration, although agreeing in the principal points, not a single one is exactly similar to any of the others in all of the particulars. In Graefe's case the facial paralysis was complete only on the left side; on the right side there was paresis. In the cases of Harlan, Chisolm and Möbins the paralysis was complete only in the upper portions of the face. In the lower portion there was some mobility about the angles of the mouth. In our case only the right corner of the mouth is mobile.

In regard to the impaired mobility of the eyes, there was in Harlan's and Chisolm's cases congenital converging strabismus. In Chisolm's case the patient had undergone a myotomy of the internal recti in early childhood. When Chisolm saw this patient she was thirty-five years of age, and her eyes were parallel. As in the other three cases, there was in those of Harlan and Chisolm a complete paralysis of the abducens on both sides. These observers have not stated clearly whether the shortened internal recti muscles responded to an impulse of accommodation. Harlan merely says that the function of all the orbital muscles but the abducent seemed normal. Chisolm states that in his case the lateral motion in both directions was absolutely impossible, because the abducens of either side was paralyzed from birth on the one hand, while on the other hand the power of the internal muscles had been completely arrested by the operation for the strabismus. One misses

² Graefe-Saemisch, *Handbuch der Augenheilkunde*, Bd. vi., p. 60; also Siebenter Periodischer Internat. Ophthalmologen-Congress, 1888, p. 30.

³ Transactions of the American Ophthalm. Society, 1881, p. 216.

⁴ Arch. of Ophthalm., edited by Knapp and Schweigger, vol. xi., p. 323 (1887).

⁵ Münchener Medicinische Wochenschrift, 1888, No. 6 (February 7).

in the last case a definite statement as to the ability to converge, especially as Chisolm found it necessary to order convex glasses for hyperopia, in order to enable her to see clearly while sewing. In the three other cases, namely, those observed by Graefe, Möbius and myself, no converging strabismus was present.

There is a remarkable similarity between Graefe's case and the one described in the beginning of this article in regard to the differences of refraction existing in the two eyes. In both cases the right eye is myopic while the left one is emmetropic. Furthermore, in both cases there is the slight vertical deviation of one eye. In Graefe's case, however, the left eye was directed a trifle lower permanently, while in our case the deviation was not constantly manifested, sometimes the right eye and at other times the left squinting upwards. In looking at an object held close to the face, this upward deviation, when existing, would always disappear in our case.

Möbius observed a difference in this respect only when the glance was directed downwards. The mobility of the eyeballs was limited in the downward movement and convergence always took place. It may be well to state here that in our case there was no abnormal limitation of downward movements, and that in fixing the gaze upon near objects in a downward direction there never is an excessive or abnormal convergence.

So far there has not been a report of an autopsy on a case where this complication of symptoms existed.

The epicanthus, the bulging glabella, the uvula bifida, the crooked index finger and the undeveloped funnel-chest are unique accompaniments, as are also the anomalies existing in the regions supplied by the trifacial and hypoglossal nerves. Möbius observed in his case a web between two fingers of the right hand.

The first observation of paralysis of the lateral motion of both eyes was made by Stellwag v. Carion.⁶ In his case,

⁶ Ueber gewisse Innervationsstörungen bei der Basedow'schen Krankheit. Wiener Med. Jahrb., Bd. xvii., p. 25 (1869). First Case.

however, the affection was not congenital. It occurred when the patient was twenty years of age, in the course of a strumous exophthalmia. The description of the earlier stage of this case corresponds essentially to the conditions found by Graefe, Möbius and myself. The axes of both eyes were parallel to each other and to the median plane, while movements to the right or left were impossible to the slightest extent. The power of convergence was fully retained. The conditions present in a subsequent stage, however, resembled rather those found by Chisolm and Harlan. Both eyes, especially the left one, were directed inwards. If the object the patient was looking at was moved out of the median plane to the right or the left, the corresponding eye only followed it until it stood parallel to the median plane, beyond which it never went, the opposite eye remaining in a strongly adducted position. Later again, the right eye returned to its normal position, while the left eye was directed a trifle to the inner side. During the course of the illness other severe complications arose, such as asthmatic attacks and mental disturbances. The limitations of the mobility of the eyes decreased constantly and the condition of the visual organs had almost returned to the normal state when the patient withdrew from observation.

A NEW MECHANICAL TREATMENT FOR TABES.

The "Times and Register," Nov. 3, 1889, reports from the "B. klin. Wochenschrift," according to Juergensen, favorable results obtained in tabes dorsalis from the application of Hessing's corsets, made from finely woven cloth and thin steel ribs. The freeing of the spine produced by wearing the corset soon results in relief from cystic and intestinal trouble; then the lancinating pains cease, and the patient walks better. The patellar reflexes are not restored. General improvement is most marked. The corset must be worn for years, under the supervision of the maker. No explanation is given by Juergensen, unless the circulation and nutrition of the cord are improved by the extension of the spine. Erb has seen favorable results in two cases from this treatment.

L. F. B.

A CASE OF MULTIPLE CEREBRAL SOFTENING,
WIDESPREAD ENDARTERITIS, DISSECTING
ANEURISM OF BRANCH OF LEFT MID-
DLE CEREBRAL ARTERY.¹

By CHRISTIAN A. HERTER, M. D.,

Physician to the Class of Nervous Diseases, Presbyterian Hospital Dispensary.

FOR the opportunity of studying the case here reported I am indebted to Dr. Andrew H. Smith, under whose charge the patient lay in the Presbyterian Hospital.

The patient, M. L. D., was thirty-two years of age, married, and a native of the United States. Her father died at an advanced age in a state of apoplexy. With this exception the family history is negative. The early history of the patient is without special interest. She has had two healthy children. One was born during the twenty-third year of her age, the other during her twenty-fourth year. Both were born at full term. Soon after the birth of the second child, the patient again became pregnant, but the pregnancy was arrested in the third month by the use of instruments, without the knowledge, it is said, of the husband. The patient made a good recovery from the abortion. No history of syphilis can be elicited from any source. The husband had no hesitation in stating that he had several times had gonorrhœa, but was certain that he had never had a chancre or secondary manifestations of syphilis. A careful examination of the patient revealed no evidences of syphilis. The possibility of syphilitic infection cannot, however, be positively excluded.

The patient has always used alcohol, in the form of beer and light wines in moderation, never in excess.

Up to the beginning of her fatal illness the patient was in ordinarily good health. She ate and slept well, and had no ailments other than constipation and an occasional headache.

On the morning of March 2, 1888, soon after waking, she

¹ Read before the New York Neurological Society, February 4, 1890.

complained of numbness of the left foot and of weakness of the left leg, which condition is said to have continued stationary during that day. On the following morning the paralysis was more obtrusive, and was observed to have extended to the left arm, which was markedly paretic. On trying to stand erect, after rising from bed, the patient fell to the floor. The leg paralysis is said to have been complete. No anæsthesia or analgesia of the arm was observed.

In the course of two or three weeks the patient gained sufficient power in the left leg to enable her to walk with the aid of a cane. Recovery of power in the arm was more rapid and complete. The patient continued to drag her leg in walking.

During the interval between the attack just described and the second seizure, the patient's general condition was good. She ate and slept well and was free from headache. It was observed, however, that her disposition was somewhat changed. She had grown irritable and capricious.

On November 5th, of the same year, seven months after the onset of the left hemiplegia, the patient experienced a new attack. Soon after she arose in the morning, the husband observed that she had some hesitation in speech; she spoke slowly and apparently with difficulty, and committed errors in the use and form of words. She was also unusually silent during the morning, but did not complain either of pain or weakness.

At three o'clock of the same day the patient became giddy and fell to the floor. She soon complained of loss of power in the right leg and arm, the face being unaffected. The mental state of the patient was dull and listless. No loss of consciousness, strictly speaking, occurred at the time of the attack. She was apathetic, but could be roused without difficulty at any time, and appeared to recognize those about her. At no time did she complain of pain or discomfort. She replied in an incoherent manner and with increased hesitation to questions put her. It is said that she never took the initiative in speaking, and never volunteered any information without being first interrogated.

On November 15th, ten days after the second attack, the patient was admitted to the Presbyterian Hospital, in a state of mental hebetude. The following notes were made soon after admission:

Examination shows the patient to be poorly nourished and anæmic. Temperature, pulse, respiration, and urine normal; urates very abundant in urine. Heart negative,

sounds feeble; lungs normal, respiratory murmur feeble; spleen normal; stomach somewhat dilated; area of liver-dullness encroached upon by area of tympanitic resonance from stomach and intestines; below free border of the ribs, on the right side, a movable boggy mass is felt, presumably fecal in nature; it is difficult to accurately determine the condition of sensibility; sensibility to pain and touch are normal; the temperature sense seems normal; the condition of the muscular sense cannot be determined; the superficial and deep reflexes are present on the left side; on the right side the knee-jerk is feeble and is obtained with difficulty; the plantar reflex is absent on this side; the abdominal and epigastric reflexes are present. The pupils are equal in size, and moderately dilated; they react during accommodation; to light they react slightly and sluggishly. Sight, taste, hearing, and smell seem normal. The hemiplegia is difficult to make out, owing to the mental state. The patient lies upon her back, with arms and legs extended, without changing her position. She sleeps a large part of the day, but is readily roused from her stupor for a few seconds. She makes no complaints and never volunteers to speak, but occasionally replies incoherently to a question. While awake the eyes are commonly fixed on some distant object. The patient is entirely indifferent to what transpires about her. Her appreciation of time and place is highly defective. Urine and fæces are passed unconsciously. There is no sphincteric weakness.

There was no material change in the condition of the patient until November 30th, two weeks after admission, when it was observed that she had a temperature of 100° .

During the remaining period of the patient's life, a period of two weeks, the temperature ranged irregularly between 101° and 104° , the average temperature growing gradually higher from day to day. There were no rigors. No physical signs of thoracic disease were at any time detected, though repeated examinations were made. There was no cough.

One week before death the pupils were observed to be somewhat more dilated than before, the left being the larger. Four days before death there appeared conjugate deviation of the eyes to the left. This condition continued, without undergoing modification, until death. Ophthalmoscopic examinations were made every second or third day during the last three weeks of the patient's life, but failed to show any changes in the fundus oculi.

About the time of the development of conjugate deviation, the left arm was observed to be in a state of catalepsy. The muscles of the arm imposed a wax-like resistance to passive movement; but this resistance was readily overcome, the arm remaining for some time in any position impressed on it. This condition was very marked in the muscles moving the elbow-joint, and continued, until the death of the patient, as a constant phenomenon. In the shoulder-muscles it was less pronounced and of shorter duration.

On the morning of December 14th the patient died in a comatose condition.

AUTOPSY.—Double hypostatic pneumonia. No cardiac disease. No renal disease. Meninges normal. Left hemisphere distinctly larger in every dimension than the right. No evidence of disease on external surface of cerebral hemispheres. Examination of the larger arteries at the



FIG. 1.

base of the brain shows no evidence of vascular disease. A number of coronal sections were made through the hemispheres at short intervals. They show the existence of several foci of softening. One of these occupies a considerable part of the left frontal lobe, as is seen in Fig. 1. (a) which roughly indicates its limits in one plane. This patch of white softenings is also seen in Fig. 2 (a), where its extent is less than in Fig. 1. It extends far forward into the frontal lobe, probably to within 1 cm. of the cortex. Posteriorly it extends about 1 cm. caudad of the plane represented in Fig. 2, but is here of small lateral extent.

Anteriorly the left half of the callosum is destroyed by the softening as far as the median line. As the lesion is followed back, the callosum is involved at a distance from the median line. A pigmented connective-tissue cicatrix is present in the right hemisphere in the situation indicated in Fig. 2, i. e., occupying the lateral part of the caudate nucleus and the mesial part of the internal capsule. This focus is of considerable antero-posterior extent (about $1\frac{1}{2}$ cm.), but occupies the lateral part of the caudate nucleus and the mesial part of the internal capsule in all the sections in which it appears. Immediately frontad and caudad of the plane shown in Fig. 2, the lesion becomes somewhat

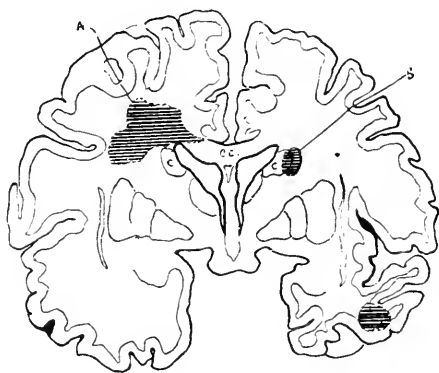


FIG. 2.

smaller. This focus undoubtedly indicates the position of an old area of softening. Another focus of softening is seen in the right temporal lobe just beneath and involving the cortex. Still another patch existed in the left occipital lobe, but its exact position was not noted. A microscopical examination of material from the large focus of softening showed it to consist of broken down brain-tissue, remains of nerve-fibres, droplets of myeline, a moderate number of red blood-cells, very few white blood-cells, compound granular corpuscles, and débris.

A number of the smaller arteries were examined carefully, both macroscopically and microscopically, among them both middle cerebral arteries and several of their branches, and both anterior cerebral arteries. Without

exception all the vessels examined were the seat of endarteritis. In some cases the increase of connective-tissue in the intima was slight in amount, in others the formation of new connective-tissue was so extensive as to seriously encroach upon the lumina of the vessels. In many sections there was a moderate degree of periarteritis. In some of the branches of the left middle cerebral artery the changes in the walls of the vessels were very advanced, more advanced than in any other arteries examined. Degenerative changes (fatty degeneration, calcification) were not observed in any vessel.

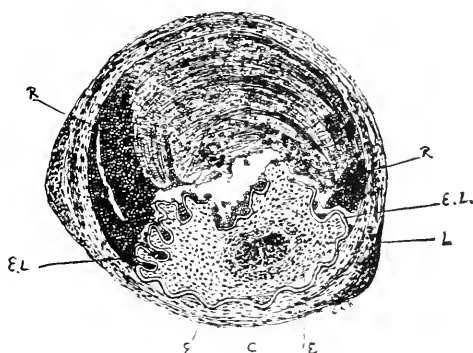


FIG. 3.

A remarkable condition was discovered in a large branch of the left middle cerebral artery. The walls of this vessel, in addition to being the seat of endarteritis, were separated by a hæmorrhage of considerable size, constituting a dissecting aneurism. The longitudinal extent of the aneurism was nearly one inch, but no accurate measurement was made. Fig. 3 represents the appearance of a transverse section of the vessel. The effusion of blood occurred between the elastic layer and the media, the former being crowded over to one side of the vessel. The effused blood which thus separated the walls of the artery occupies a large area. On either side of the space occupied by the hæmorrhage large numbers of red blood-cells are seen heaped together (in the drawing the cells are represented disproportionately large). In the remaining space

the effused blood shows signs of organization. The area is transversed by numerous streaks of fibrin forming a fine meshed network. A high power shows these meshes to be filled with large numbers of red blood-cells, which have lost their color and lie massed together. In the loops formed by the compression of the elastic layer, lie imprisoned many red cells. The intima is the seat of an endarteritis of considerable intensity. The lumen of the vessel is occluded by a small thrombus, apparently of the same age as the large clot. The vessel is the seat of a moderate degree of periarteritis. No condition similar to that just described was found in any other vessel. Neither was the lumen of any other vessel examined completely obliterated. Unfortunately, the exact position of the vessel which was the seat of the dissecting aneurism was not carefully observed, the relations having been lost before it was discovered that the artery was of especial interest.

Observations.—No accurate diagnosis of the nature (softening) of the cerebral lesions above described was made during the life of the patient, as nothing whatever was known of her history before admission to the hospital until the time of her death, when the facts incorporated in the clinical history were brought to light. As the patient could give no account of herself, the diagnosis rested entirely upon the objective features of the case. For a considerable period the mental defect and right hemiplegia were the only evidence of an intracranial process. The lesion causing the hemiplegia was thought to be located in the internal capsule, or in the motor path between the capsule and the cortex, and the grave mental failure was referred to a destructive process involving one or both frontal lobes. As to the pathological character of the lesion, no opinion was hazarded. Hæmorrhage was considered improbable, because of the age of the patient and the absence of the causal indications of hæmorrhage—atheroma of accessible arteries and Bright's disease. The absence of arterial and renal disease made softening from atheromatous thrombosis improbable, and there was nothing to suggest embolic softening. Other forms of softening were not seriously considered.

Abscess was thought of, but could not be regarded as probable. The absence both of irritative phenomena and optic neuritis made it unlikely that the symptoms could be referred to a new growth. Viewed in the light afforded by a study of the lesions found in this case, there are two clinical features that require comment—the high grade of mental defect observed, and the existence of catalepsy. The mental defect can undoubtedly be referred in part to the destruction of a considerable portion of the left frontal lobe, as the lesions in other parts of the cerebrum are of small extent. But the mental failure seems entirely out of proportion to the extent of destruction in the frontal lobe. The corpus callosum was softened at its anterior extremity, and it is not improbable that the destruction of this important commissural tract is responsible for a degree of mental loss that cannot be accounted for by the mere extent of the lesion in the frontal lobe.

The existence of localized catalepsy is of interest in connection with the lesion observed in the right internal capsule and caudate nucleus. This focus, which is older than the larger lesion on the left side, is unquestionably related to the first hemiplegic seizure noted in the clinical history. Perhaps it is also related to the catalepsy, but no positive expression of opinion can be ventured on this point. The fact that the catalepsy was localized and not general in its distribution makes it reasonable to suppose that it is dependent on the focal lesion described rather than on the mental state, but only future observations can determine whether this symptom ever possesses localizing value.

But by far the greatest interest of this case lies in the peculiar form of arterial disease that has been described. A dissecting aneurism led to the perfect occlusion of an artery of considerable size. This artery was an important branch of the left middle cerebral. Unfortunately, its exact location was not noted, and no positive statement can be made with regard to the area supplied by it. Yet the fact that it was completely occluded and that none of the other vessels examined were so occluded, makes it

highly probable that the aneurism was operative in the production of the softening, though possibly not of the entire area. The time at which the second attack of hemiplegia occurred corresponds closely with the appearance of the clot, and there can be no reasonable doubt that there is a direct causal relation between the intra-arterial hæmorrhage and the softening in the motor path to which the paralysis is due.

In regard to the origin of the dissecting aneurism there is little to be said. Possibly it is related to the pre-existing endarteritis, but it is certainly an exceedingly rare consequence or association of such endarteritis. In the literature at my disposal I have been unable to find a description of any condition resembling even remotely that which I have described.

The nature of the endarteritis is also obscure. It is true that it involves chiefly the smaller vessels, and that no degenerative changes can be detected in the vascular walls, and such endarteritis is usually looked upon as rather characteristic of a syphilitic origin. Possibly the endarteritis is of syphilitic origin, notwithstanding the improbability of syphilitic infection in this case. I believe that many cases of endarteritis are set down as syphilitic where such origin is highly improbable, and that future research will bring to light causes of endarteritis at present not appreciated. In my experience it is a prevalent belief that cerebral softening is not infrequently a primary affection, occurring without the existence of vascular disease. Probably one reason for this belief is that it is not uncommon to examine the cerebral arteries in cases of softening in an imperfect manner, arterial disease being excluded because the important vessels at the base appear normal. If the presence of vascular disease were excluded only after a careful examination of the vessels, large and small, I believe that primary cerebral softening would no longer be recognized.

A CONTRIBUTION TO THE STUDY OF EPILEPSY.

By FRANK H. INGRAM, M. D.

Pathologist the N. Y. City Asylums for the Insane; Visiting Physician to the Hospital for Nervous Diseases.

IN observing a large number of cases of epilepsy for a long period, one is necessarily impressed by the various phases which the disease manifests in one person, and by the difference in the character and in the sequence of the convulsive phenomena in different persons. Viewed from a purely clinical stand-point, it would seem a comparatively easy matter to so group the cases that a series of types of epilepsy could be definitely formed, and an instructive lesson in treatment and prognosis be acquired. It was with the hope of establishing some basis for the selection of cases for treatment that the observations on which this paper is founded were made. The results of months of patient work was far from gratifying in many respects, but sufficient information was gained to justify the trouble expended.

In the cases herewith presented, the time during which observations were made and recorded varied from eight months to several years. Ninety of these cases were so located that they could be conveniently grouped for statistical purposes and comparison, while the others were selected for the special features of their disease. I am indebted to Dr. Charles C. Flint, of Lenox, Mass., for the compilation of the table showing daily movement in ninety cases during a period of eight months. This time really represented a year, for in the four months remaining nothing of an exceptional nature occurred to qualify the record. The disease had been established for not less than one year in any case, and in a few cases it had existed upwards of twenty years.

During the period mentioned, 110 epileptics had a few more than 11,000 distinct epileptic seizures. These were,

in the main, well defined convulsions; a few were vertiginous attacks of mild form, without spasms of the limbs; others were pure motor explosions of a non-convulsive type. A good majority of these seizures occurred during the day, as will be seen by reference to the following record of ninety cases:

Month.	May	June	July	Aug.	Sept.	Oct	Nov.	Dec.	Total.
Diurnal.....	738	1042	666	726	793	956	843	886	5750
Nocturnal....	530	855	475	561	502	444	352	447	4106
Total.....	1268	1897	1141	1227	1295	1400	1195	1333	9856

Here we have a total of 9,856 epileptic seizures occurring during a period of eight months. Within this time, five of the patients died or were discharged from care, so that at all times there was an average of eighty-five under observation. It will be noticed that the monthly average was 1,232, 718.75 diurnal, and 513.25 nocturnal seizures; for each patient, 14.495, 8.547 diurnal, and 6.038 nocturnal. In June the average was much higher, being 22.31+ for each patient, 12.25+ diurnal, and 10.05+ nocturnal; and the difference between the diurnal and nocturnal seizures was less than in any other month. It is an interesting fact that during this month there were many marked thermometric and barometric changes: hot days would be followed by exceptionally cold nights, and the transitions from dry to wet weather were remarkable. It seems that these atmospheric variations must have had a prominent influence in producing the excess of convulsions; and in the only other month in which the number of seizures was above the average, namely, October, there was one week of similar conditions. Sudden barometric changes invariably caused an increase in the number of convulsions, and before and after a thunder storm this was excessive. Changes of temperature did not exert such a marked influence.

The excess of diurnal over nocturnal fits may be accounted for by the fact that at night many causes of excite-

ment are absent. The external influences which affect the emotions are wanting, and the repose of the body is unfavorable to the development of motor spasms. With the exception of the one month in which the many nocturnal seizures were charged to the account of atmospheric influences, undigested food, retained urine, and bad dreams were I believe, responsible for a vast majority of the fits. I have frequently noticed that a convulsion coming closely upon the evening meal, the patient being soon put in bed, would be followed by one or more convulsions during the night; and, in such cases, it was a convenient inference that the first explosion retarded gastric digestion and that the presence of food in the stomach was sufficiently irritating to renew the spasms.

With the urine of several hours of excretion in the bladder, a fit at the time of, or soon after, retiring, would be followed by others before the night had passed. In the same cases, when the urine had been voided before the fit, a tranquil repose would, as a rule, mark the night.

Disturbing dreams were not impotent so far as the seizures were concerned. It was often observed that a patient would awaken with a start, and almost immediately after have a well defined convulsion. In these instances the circumstances were such as to exclude the probability that the startled awakening from sleep was the beginning of the epileptic seizure. With the more demented patients, constipation and retention of urine were habitual, so that two exciting causes of convulsion were ever present.

The aura epileptica was defined in less than one-half of the cases under observation. More frequently the fit was sudden and ushered in by loss of consciousness, the spasms of the limbs following after an appreciable interval.

The stage of convulsion varied in duration from thirty seconds to as many minutes. The greatest violence was, as a rule, manifested at the middle of the attack, although sometimes most prominent at the beginning, or at the termination, of the spasms.

The periods of coma or stupor lasted from a few minutes to several hours. Occasionally there would be a series of

apparently voluntary, but decidedly irrational, acts before consciousness was regained. This is well illustrated in two cases, as follows :

A man, twenty-five years of age, had had epileptic seizures with varying frequency during a period of ten or eleven years. As a boy of from thirteen to fifteen years, he masturbated almost incessantly, not because of a developing sexual desire, but for the reason that there was a peculiar irritation of the genitals, which was relieved only by friction of the parts. His foreskin was long and adherent, and circumcision was performed, with some benefit to the sufferer. The epileptic seizures in this case were of the ordinary kind up the time of emergence from stupor. At the period when consciousness should have returned, this man would arise from where he had been placed, would wash his face and hands and carefully adjust his disordered garments, then, with gravity of countenance and dignity of demeanor, perform ludicrous but pitiable acts. He would disarrange the furniture and ornaments of his room, or would urinate in a vase or between the mattresses of his own or a fellow-patient's bed. His appearance indicated consciousness, but he had no recollection of these acts, and a careful observation of the case demonstrated that he was not conscious when performing them. Some months this patient would have five or ten fits and fifteen or twenty nocturnal emissions of semen ; again, this order would be reversed. There was ever a definite relation between the fits and the emissions, and the latter probably represented localized epileptic spasms.

The second patient of this class, a man twenty-one years of age, had had epilepsy for eight years. His seizures consisted of, in the order named, stupor, rolling of the body from side to side, with mild spasms of the limbs ; somnolence ; extreme terror, accompanied by violence in action ; exhaustion and gradual return of consciousness. During the period of terror, he would run at highest speed for a mile or two miles, vaulting fences and other obstructions with an unnatural and surprising agility ; or he would expend his violence in tearing boards from their fastenings

or in chopping wood with an axe. The stage of exhaustion would last ten or fifteen minutes, during which the patient would stagger about in a feeble way, or he would seat himself or lie down in some convenient place. Consciousness returned slowly.

Another case, in some respects similar to the two just described, was an unmarried woman, twenty-five years of age. Frequently she would have the most common form of epileptic seizure, but fully as often her disease would be manifested in unconscious acts of extreme violence, without convulsions, which would be succeeded by somnolence or stupor. She did not remember what had occurred during her paroxysms of violence.

A case deserving at least a passing notice was a woman thirty-eight years of age. Her epilepsy consisted of infrequent nocturnal convulsions, always attributable to an undigested supper, and of frequent attacks of *petit mal* or vertigo. The vertiginous attack was a simple, momentary loss of consciousness; the other form of seizure consisted of loss of consciousness, accompanied by incoherent mutterings, which would last from thirty seconds to three minutes. In these there would be apparently but a suspension of continuity of mental action, for, no matter whether, at the onset, the patient had been reading, knitting or engaged in conversation, with the return of consciousness the train of thought would be taken up at the broken point and carried out in orderly sequence. This one feature, of itself, makes a conspicuous departure from the usual form of attack of *petit mal*. This patient was anæmic and suffered from constipation and hemorrhoids. Tonic treatment and ligation of the piles caused a most marked improvement, but a cure was not effected. In this case there was a partial amnesia, which, in degree, was disproportionately more severe than the fits.

The most peculiar character of convulsion witnessed in any case was a combination of almost every conceivable motion of the body and limbs. After a cry and a twisting of the body, the patient, a woman, twenty-three years of age, would fall to the floor; the top of the head would be

fixed as a pivot; the body would rotate and, at the same time, describe a circle with its length as the radius; the limbs would engage in a series of clonic convulsions of greatest severity, and the fit would terminate in a tonic rigidity of all the muscles, which would be followed by relaxation and stupor. Exhaustion was not as marked in this case as might have been expected, considering the violence of the explosion, and the mind cleared quickly.

I have the records of three autopsies in which the changes in the brain would fully account for the epileptic convulsions. These are given with such parts of the histories of the cases as are pertinent.

CASE I.—A woman, forty-five years of age, had been afflicted with epilepsy for more than ten years. The seizures were frequent, often as many as six occurring in a day. Towards the end of life the fits were most severe, and death occurred from exhaustion a short time after emergence from the epileptic state. The fits were invariably of a kind: spasms of the left leg, spasms of the arm of the same side, loss of consciousness and convulsions of the limbs of both sides. For a short period after each fit, the left side appeared weaker than the right side.

The mental state of this woman had a history not uncommon in epilepsy. Some three years after the development of the disease, she manifested considerable irritability and had frequent outbursts of marked excitement. Eventually the excited periods increased and a well-defined mania developed. Paroxysms of violence were frequent and delusions were wanting. As years passed, excitement was less marked and the mind became sluggish, but dementia was not complete.

The post-mortem examination showed a poorly nourished body. The heart-walls were thin and the ventricles dilated, but there was no evidence of disease of the lungs or of the abdominal viscera. In the right hemisphere of the brain, the subcortical vessels of the "leg-centre" were dilated and tortuous, presenting the appearance of multiple aneurisms. This condition, so much in keeping with that of the heart, was found in no other part of the brain, and it will, I believe, fully account for the peculiar character, as well as for the existence, of the convulsions.

CASE II.—A man, thirty-two years of age, was suddenly seized with loss of consciousness and paralysis of the left side of the face and body. The recovery from the hemiplegia was rapid, but there was a subsequent deafness and a paresis of the face and tongue on the left side, and epilepsy soon developed. The convulsions always began, were more violent and of longer duration on the left side. Death terminated the disease, after twenty-four hours of convulsions and coma, in the fourth year.

The post-mortem examination of the brain showed deposits of caseous matter, in the form of granules, in the sheath of each auditory nerve and between the skull and dura and between the dura and pia, in the right Rolandic region. The pia was, in this area, fixed to the brain by fibrinous and caseous matter, and the blood vessels were coated with similar substances. There was a slight cortical degeneration, probably a softening caused by pressure of the deposits, but the other parts of the brain were normal.

CASE III.—The history of this case is unimportant, excepting that the convulsions were synchronous on the two sides of the body. The autopsy revealed an area of degeneration in the posterior horn of each lateral ventricle. This is the only case in my experience, although much care has been taken in many examinations, in which there was an indication of lesion in the so-called epileptogenic zone.

When we view epilepsy in its various phases, observe the many ways in which it manifests itself as to the character of the convulsions, and note the different findings in *post mortem* examinations of the brain, it would not be reasonable to expect to discover a definite pathology for this disease. Cortical and ventricular degenerations, various kinds of deposits, varicose and aneurismal vessels will be found singly or in combination. Various kinds of disorders of nutrition and vascular weaknesses are frequently the only apparent defects in cases of epilepsy. Psychic influences are most potent in generating the explosions.

In conclusion, a few words in regard to the treatment of epilepsy. The patients of whom I have written have been under medical care at all times, some in hospitals and others at home. About one-half took bromides; the remaining ones took various other drugs. The bromides in some instance greatly reduced the number of convulsions,

but nitro-glycerine, iron, digitalis, and other drugs did almost as much good. The bromides seemed especially adapted to some cases, but their ultimate effects were frequently so undesirable as to materially affect the value of the drug.

The tendency of epilepsy is toward dementia, more or less complete. It would, therefore, seem fitting that the physician should select remedies which will correct any functional disturbance in the circulatory, digestive or urinary systems, and that he should avoid, unless particularly indicated or absolutely demanded, those restraining remedies which, long continued, impoverish the blood and hasten mental degeneracy.

ANTIPYRIN IN ANGINA PECTORIS.

The "Epitome" (Dec., 1889) notes the good effects of this remedy in severe angina pectoris. The patient was brought into the ward unconscious, morphine and trinitrine having been administered. When Dr. E. D. Martin (New Orleans) first saw him, he was undergoing much agonizing pain, that five drops of nitrite of amyl (by inhalation) magically relieved. Large doses of antipyrin and digitalis were ordered three times daily, with a view of diminishing the reflex functions. On the third day the antipyrin was stopped; but on the fourth pain returned, and the same treatment was again employed for ten days, with flattering results. A striking feature of the report is the patient's own intelligent history of the case, the procedure instituted by Dr. Martin for his relief corresponding exactly to his own method of diminishing torture and one that he had followed for five years. Earlier, he had used chloral during, and bromide of potassium after, the attack. There existed strong hereditary predisposition to angina pectoris, all of the patient's family suffering more or less from the same trouble.

L. F. B.

REPORTS OF SIX CASES OF FRIEDREICH'S ATAXIA, OCCURRING IN THREE DIFFERENT FAMILIES.

BY DR. CHARLES W. ROOK, OF QUINCY, ILL., AND DR. CHARLES L. DANA, OF NEW YORK.

THE histories of the first four cases were sent to me, with photographs, by Dr. Rook. The histories given leave no doubt in my mind that they are true cases of Friedreich's ataxia.

To them I have added one case, recently observed by me, and a second, which was sent to me by Dr. J. W. S. Gouley, and which has been under my observation for several years. This latter patient's history was reported in brief in the "Medical Record," of October 1, 1887. It will appear soon, in full, in an article on Friedreich's ataxia, by myself, in Keating's "Cyclopædia of Children's Diseases." I therefore omit the publication of the case here. Some comments upon the disease, and some new observations made upon my cases, have been embodied in the article referred to, and cannot properly be published now.

I find that there have now been reported about 165 cases. Fifty-four, or one-third of them, have been reported by seventeen different American observers. I conclude that Friedreich's Ataxia is a relatively frequent disease in this country.

C. L. D.

HISTORY OF DR. DANA'S CASES.

CASE I.—B. N., United States, aged twelve. One paternal uncle had some spinal trouble; one paternal aunt had some spinal trouble, and wore braces from tenth to forty-fifth year; father had some spinal trouble, and walked ataxic in last ten years of life, when he died of pneumonia. Patient is youngest of four children: one boy, twenty-four; one boy, twenty; one girl, fifteen—all of whom are well.

Birth natural, at full term. He seemed well until his seventh year, when he had scarlet fever. Has been in poorer health since, but no especial trouble was noted until the present one developed. No history of diphtheria. He had whooping-cough in the spring of 1888. In the following winter of 1888-89 he was noticed to stumble in his gait while playing, and especially to stumble in the dark. His gait was awkward and irregular. He complained of no pain in the legs, but had some pains in head and stomach occasionally. No bladder or rectal troubles, and no disorders of vision or speech were complained of.

He was sent to Dr. S. N. Phelps, hip-disease being suspected. Dr. Phelps excluded any such trouble, and referred him to me, October 16, 1889.

When examined by me he showed a gait somewhat ataxic and stumbling, but also with an apparent limp in the right leg. He could not walk far with the eyes closed, nor could he stand with the eyes closed and the feet together except for a short time. He was well grown, the legs being a little small, but not showing any local atrophy or hypertrophy. His speech was somewhat peculiar; but this it had always been. No nystagmus; vision and optic nerves normal. His knee-jerks were present. Slight tactile anæsthesia over right foot, and dorsal flexion of right great toe were noted. Urine normal; electrical reactions normal.

He had no pain; no spinal curvature. Essentially his trouble was only an ataxic gait; the arms not being affected. This ataxia was very apparent, however, and was worse in the dark; so that though clumsy in gait at all times, he was especially so at night.

The case is in its incipient stage, and all the symptoms are not present; but, in view of the dominance of ataxia and the family history, there seems to be no doubt of the correctness of the diagnosis.

CASE IV.—J. D., aged twenty-one, United States. No family history of ataxia or other nervous disease. It developed at the age of fifteen, after a blow on the head, and was associated with polyuria and speech-disturbances. The ataxia and the peculiar rolling gait are very marked.

HISTORY OF DR. ROOK'S CASES.

Their maternal grandfather died of phthisis at the age of seventy years. Their grandmother is sixty-eight years old, and enjoys good health. To them were born eight children. One son died in convulsions when nine months

old, and another of phthisis at the age of twenty-two years. Three sons, in good health, with families in which no ataxia has developed. The three daughters have good health. One is single; one is married seven years, but has no children; and one, the mother of my cases. She is of medium build, and forty-three years old. She has had no serious sickness. Intoxicants, chiefly beer, are employed as a beverage in the family of the latter.

In the family history there are no instances of consanguinity, syphilis, insanity, or ataxia. The only predisposing causes revealed are phthisis and alcoholism.

To Mr. and Mrs. G., parents of my cases, were born eight children, as follows:

1st. Julia, aged twenty years, ataxic since the age of eleven years.

2d. Laurence, aged eighteen years, not ataxic.

3d. Antone, aged sixteen years, ataxic since the age of eleven years.

4th. Clara, aged fourteen years, ataxic since the age of eleven years.

5th. Katie, aged twelve years, not ataxic.

6th. Bertha, aged nine years, ataxia developing.

7th. Infant daughter, died of whooping-cough at the age of sixteen months.

8th. Infant daughter, died of inanition at the age of seventeen months.

CASE I.—Julia G., aged twenty years. Her early life was very free of sickness. In her second year she had some fever during dentition; and in her eleventh year an attack of measles, from which she recovered without complications.

During her eleventh year she first experienced a sense of weakness in the lower limbs, and in a few months her gait became staggering. These symptoms gradually increased till she was unable to walk or stand without support. Her arms were affected one year later, or during her twelfth year. Since the commencement of this disease she has had no other sickness.

She is now much deformed by contractures. There exists a kyphosis, a left dorsal and a right lumbar scoliosis, a double talipes valgus; when at rest, the hands assume a wrist-drop and the fingers a claw-like appearance.

She is not well nourished, though the food taken is digested without distress. Physical examination of lungs and heart negative. Respiration 18 and the pulse 80 per minute. Urine, reaction acid, sp. gr. 1.020, and contained no morbid elements.

The mammæ are partially developed, and the menses have thrice occurred, at intervals of two or three months, during this her twentieth year.

The expressionless appearance of her face is less marked when attentively listening to or engaging in conversation. When reading she pronounces slowly yet distinctly, but in conversation her speech is slower, voice more tremulous, and acts of cerebation are performed with an effort. She has a good memory, as shown by the fact that she can yet instruct her brothers and sisters in reading and mathematics, though it is eight years since she was at school.

Her sleep is natural in appearance, though she requires from nine to fourteen hours daily. Her disposition is becoming more irritable than in the earlier years of her affliction.

Decided atrophic changes have occurred throughout the entire muscular system. No muscles are paralyzed, though their electrical reaction is much less than normal.

Co-ordination is very deficient, more so in the lower than upper extremities. Though unable to stand or walk without support, ataxia of station is probably increased by closure of her eyes, for then the movements of her arms become more unsteady.

She is able to feed and dress herself and perform some work, as the washing and drying of dishes and sewing. Several minutes are required for her to thread her needle, and then only by supporting one hand with the other.

She has some attacks of vertigo, usually soon after rising of a morning or after meals. She often experiences a feeling of numbness in her limbs, but no pain.

Cutaneous sensibility is diminished, particularly in the lower limbs, where two points of pressure may be separated as much as four inches and described as only one point of pressure.

Her vision is good, no nystagmus, and ophthalmoscopic examination of the eyes negative.

The sense of hearing, taste, and smell normal. Superficial reflexes, as the plantar, are diminished. Ankle clonus and patellar reflexes absent.

The extremities are colder than natural, but not œdematous. No secretory disturbances, save that of the menses.

CASE II.—Antone G., aged sixteen years. When seven years old he had measles, from which he recovered without complications. During his eleventh year his parents first noticed his staggering gait, and about one year later his arms became affected.

There now exist no contractures except a marked kyphosis, which can be partially overcome at will, and a double talipes valgus.

His gait is very ataxic, the feet being separated some six or eight inches in order to better maintain his equilibrium while standing or walking.

Over slight obstructions he stumbles, occasionally falling; yet, notwithstanding this difficulty, he has, during this year, taken many walks of two or three miles.

His nutrition is good, and the functions of the primæ viæ are normal.

Physical examination of lungs and heart negative, though he has frequent attacks of palpitation.

On three occasions were witnessed attacks of syncope that are worthy of record. The first attack occurred while in the Sayre suspension-apparatus; second, while standing and being examined for spinal curvature; and third, while sitting and being examined to determine the electrical reaction of the muscles.

The strength of current employed, that induced the last attack, was only sufficient to produce, with the electrodes placed on the right arm, at elbow and wrist, very slight flexion of the hand.

He would announce the onset of the attacks by the remark, "I feel so dizzy." His head would sway from side to side, face become pale, radial pulse disappear, muscles relax, and unconsciousness supervene.

The relaxed condition would continue for possibly half a minute, during which time the pulse and respiration were not perceptible; then followed a sudden and violent tonic convulsion, his body assuming a position of extreme opisthotonos. In about one minute the convulsive state began to relax, consciousness returned, and for some minutes he suffered severe pain in the erector spinæ muscles. During the seizure the pulse and respiration returned, the latter being stertorous, and the kyphosis completely disappeared.

Temperature is normal, respiration 19, and pulse 84.

The urine is acid in reaction, specific gravity 1.025, and contained no morbid elements.

His intellect is but little affected, memory good, speech slow, voice tremulous, and facial expression habitually gloomy.

His sleep is natural, save when occasionally disturbed by spasmodic contractions of the lower limbs. These contractions are painless.

No paralysis or atrophy exists. Electrical reaction of muscles diminished.

His power of co-ordination is much less than normal; yet, under direct supervision of sight, he has fair control of his extremities. With eyes closed, the ataxia is more apparent, for then he can neither stand nor walk without support.

He has frequent attacks of vertigo, always preceding attacks of syncope. He experiences no pain, save after the convulsive seizures, but often has a feeling of numbness in the extremities.

Cutaneous sensibility less than normal; more noticeable in lower extremities, where he cannot distinguish between two points of pressure, if separated three or four inches; nor can he correctly locate a point of pressure.

Vision is good, and ophthalmoscopic examination of the eyes negative.

Sense of hearing, taste, and smell normal.

Plantar reflex diminished. Ankle clonus and patellar reflex absent.

The only secretory disturbance has been an occasional incontinence of urine.

There are no vaso-motor changes.

CASE III.—Clara G., aged fourteen years. When two years old she had an attack of pneumonia, and has since remained delicate. At five years of age she had measles, and recovered without complications.

During her eleventh year the ataxia began in the lower limbs, and within a year the arms were also affected.

The first objective symptom noticed was her staggering gait; she also early experienced a sense of weakness in the limbs.

Her gait is now very ataxic, as are also the movements of her arms. In passing through a room she will touch one or more pieces of furniture, thereby enabling her to better maintain her equilibrium.

There exist some contractures, as a kyphosis, double talipes valgus, and wrist-drop, all of which can be partially overcome.

She has a dry, hacking cough, and auscultation reveals the presence of numerous crepitant rales in each lung, but more abundant in the posterior part of the lower lobes.

The heart's action is very irritable, hastily crossing a room causing palpitation for several minutes.

Her temperature, as shown by a thermometer, is usually above normal, though the extremities feel colder than natural. There is frequent flushing of one or both cheeks. This increase of temperature and hectic is due to the diseased condition of the lungs.

Respiration 21, pulse 90.

Urine is acid, with a specific gravity of 1.024, and in other respects normal.

Her facial expression is more intelligent than her sister Julia's or brother Antone's, for it is not melancholic.

She learns easily, has a good memory; speech slow and voice very tremulous.

Sleep is natural.

Co-ordination very much diminished. She cannot walk in a straight line with eyes open, or stand with feet together without her body swaying, and will fall, if not supported, when her eyes are closed.

The ataxia is nearly as marked in the upper as in the lower extremities. When extending the hand to grasp an object, its claw-like appearance is quite noticeable.

Electrical reaction of muscles diminished. Atrophic changes are marked throughout the muscular system.

No muscle or group of muscles is paralyzed.

She has frequent attacks of vertigo, and is occasionally disturbed by a feeling of numbness in the lower limbs, but has no pain.

Cutaneous sensibility is diminished. Not able to locate or distinguish between two points of pressure any better than her sister or brother.

Ophthalmoscopic examination of her eyes negative. Vision is good. Some months ago nystagmus was quite marked; now it is only occasionally observed.

Her sense of hearing, taste, and smell normal.

Plantar reflex diminished. Ankle clonus and patellar reflex are absent.

There are no secretory disturbances.

CASE IV.—Bertha G., aged nine years. At the time of her birth the other children of the family were sick with the measles, and nine days later a cough developed and a slight eruption appeared.

She was supposed to have had the measles. When one and a half years old she had convulsions, and once or twice yearly the attacks recurred till she was six years old. The

later attacks are known to be due to indigestion, and probably the earlier ones also.

When she was seven years of age she had the measles, there being at that time an epidemic of this disease; but no other member of the family was attacked.

A slight deafness was observed after her recovery; and two months later there suddenly appeared, at each ear, a profuse otorrhœa, which ceased in a few weeks, leaving her almost entirely deaf.

She is now in good health, there being no derangement of the digestion, heart, lungs, or kidneys.

Her disposition is pleasant, and her face bright and intelligent. Speech is slow, but not tremulous. Sleep is natural.

Ataxia of locomotion or station is not apparent with eyes open, but with them closed her gait and station are each unsteady.

In the excitement of play, or when eyes are closed, the motion of her arms is also slightly ataxic.

There are no muscles atrophied, contracted, or paralyzed. Electrical reaction of muscles normal. Tactile sensation diminished. Vision, taste, and smell normal, but hearing destroyed.

Plantar reflex nearly normal. Ankle clonus absent, and patellar reflex present, but greatly diminished.

There are no vaso-motor or secretory disturbances.

The treatment employed for these patients has been, for each: silver nitrate, one grain, in pill, twice daily; for Cases I. and III., cod-liver oil in emulsion, and, for all, suspension. For six months an average of two suspensions per week have been given.

Results of Treatment.—The general health of Cases I. and III. are improved, but no change noted in their ataxia. Case II. is improved in his gait, but not in his reflexes or tactile sense. Case IV.: Ataxia not increased during six months' treatment.

REPORT OF THE COMMITTEE OF THE NEW
YORK NEUROLOGICAL SOCIETY UPON THE
GALLUP LUNACY BILL.¹

Mr. President :

The Committee, appointed at the last meeting of the Neurological Society to examine the proposed new lunacy law, known as the Gallup Bill, beg leave to report as follows :

The measure has a large number of very excellent features, which, if they should become law, would prove of the highest advantage to the unfortunates whom they are intended to benefit. These are the sections relating to

1. The removal of the insane to asylums by attendants of the same sex.

2. The admission and discharge of voluntary patients.

3. The admission of emergency cases without papers of any kind for three days.

4. The forbidding of the confinement of insane persons in jails in the same room with criminals, and any detention beyond ten days.

5. The provision of home visits of indefinite duration at the discretion of the medical officers of the asylums.

6. The boarding-out of suitable chronic lunatics in private families, at county expense, according to the systems in vogue in Scotland and Massachusetts.

Aside from the invaluable particulars just described, there are several sections relating to commitment to which there would seem to be serious objections.

In order to more fully comprehend the changes that it is proposed to make in the present law regarding this pro-

¹ Presented at the Meeting of February 4, 1890.

cedure, the law now in force, dating from 1874 (chap. 446), is here given, together with the most important sections of the Gallup Bill relating to the same matter :

Laws of 1874.

" TITLE I, § 1. No person shall be committed to or confined as a patient in any asylum, public or private, or in any institution, home or retreat for the care and treatment of the insane, except upon the certificate of two physicians, under oath, setting forth the insanity of such person. But no person shall be held in confinement in any such asylum for more than five days, unless within that time such certificate be approved by a judge or justice of a court of record of the county or district in which the alleged lunatic resides, and said judge or justice may institute inquiry and take proofs as to any alleged lunacy before approving or disapproving of such certificate, and said judge or justice may, in his discretion, call a jury in each case to determine the question of lunacy.

" § 2. It shall not be lawful for any physician to certify to the insanity of any person for the purpose of securing his commitment to an asylum, unless said physician be of reputable character, a graduate of some incorporated medical college, a permanent resident of the State, and shall have been in the actual practice of his profession for at least three years, and such qualifications shall be certified to by a judge of any court of record. No certificate of insanity shall be made except after a personal examination of the party alleged to be insane, and according to forms prescribed by the State Commissioner in Lunacy, and every such certificate shall bear date of not more than ten days prior to such commitment.

" § 3. It shall not be lawful for any physician to certify to the insanity of any person for the purpose of committing him to any asylum of which the said physician is either the superintendent, proprietor, an officer, or a regular professional attendant therein."

The Gallup Bill.

" § 3. Whenever a justice of the peace or a superintendent of the poor or a judge of a court of record shall receive information that a certain person, deemed insane, should be placed in custody, for either of the causes stated in section first of this act (Form B), the said justice or superintendent or judge shall, by an order in writing (Form C), direct two

examiners in lunacy to examine the alleged insane person and report to him within one day, exclusive of Sunday, after their respective examinations (Form D) the results of such examination, with their recommendation as to the special action necessary to be taken in the case; if a justice of the peace, or a superintendent of the poor, issues the order for an examination, he must personally visit the alleged insane person; if the physicians certify that the person so examined is not insane, the justice or superintendent shall dismiss the case, but if they certify that he is insane, and a proper subject for commitment, as provided in section one of this act, said justice or superintendent shall certify, under his hand, to the correctness of the proceedings and to his personal visit (Form E), and shall cause said certificates to be delivered to a judge of a court of record within two days, exclusive of Sunday, of the date of the last certificate made, which last certificate must not bear date of more than two days of the first certificate.

“§ 4. On receiving said certificates from a justice of the peace or superintendent of the poor, or on receiving the certificates of two examiners in lunacy appointed by himself, certifying to the insanity of any person, and recommending that he be placed in custody for cause, then and in either case the said judge may or may not visit the alleged insane person, or require that he be brought into court, but he shall state in the order of commitment whether or not he saw him, and if he did not see him he shall give the reason therefor; the judge may or may not take further testimony, and he may call a jury, but in either case, if satisfied that the person is insane, and that the reason given for his commitment in the certificates are just and right, he shall make an order (Form F), committing said person to the custody of the superintendent of the proper State asylum for the insane, or the keeper or superintendent of a private asylum or licensed house for the insane; said order shall be issued within five days after the date of the last medical certificate; a copy of said medical certificates and answers, obtained in accordance with Form D, shall be transmitted with the order of commitment to the superintendent of the respective asylum, and the originals thereof shall be filed in the office of the clerk of the county, and shall be inaccessible except on the written order of a judge of a court of record; nothing in this section shall be construed to prevent the commitment of an insane person, in accordance with the provisions of this act, to the asylum of any county authorized by law to have the care of the acute and chronic insane,

provided the said insane person is a legal resident of said county.

"§ 5. It shall be the duty of the judge, before he makes the order of commitment, to cause the alleged insane person to be fully informed of the action about to be taken concerning him; and if said insane person, or his friends or relatives, demand that other testimony be taken, or that a jury be called, the judge shall act at his discretion, but if he deny the motion, he shall state the reasons therefor in the commitment."

To present a clearer view of all of the practical differences in the two methods of commitment, they are condensed and paralleled for comparison, as follows :

PRESENT LAW.

The family physician calls in another physician. Together they make out two medical certificates and swear to them before a notary.

(This is all sufficient for admission to the asylum for five days.)

A judge of a court of record must write his name and the word approved on the back of the certificates to make them valid beyond the five days.

The papers must not be over ten days old when the patient is admitted.

THE PROPOSED LAW.

The family physician makes out a formal paper notifying a judge, justice of the peace or superintendent of the poor that a patient of his is insane and a proper subject for an asylum.

The official thus notified fills out two blank forms directing two physicians to examine the patient.

The physicians make out two medical certificates, which are returned to said official.

If the officer notified be a justice of the peace or superintendent of the poor, he must himself also visit the patient and satisfy himself as to his insanity, after receiving the certificates of the physicians. He then makes out himself a corroborating certificate, and presents the three certificates to a judge of a court of record.

The judge then sends notice to the patient of the proceedings, makes out an order committing him to the asylum, and finally issues a warrant to the asylum superintendent to send for the patient, or to a county official to remove him thither.

The judge must also cause copies of the medical papers to be filed in the office of the county clerk; and he must furthermore take proof as to the estate of the patient, filing another certificate as to these facts with the county clerk.

The process is only completed after a formal paper has been made out by the asylum superintendent to the judge committing the patient, notifying him of the admission of the case. The judge must cause this paper also to be filed with the other papers in the office of the county clerk.

At present two papers only are necessary for the commitment of a patient to a hospital for the insane. According to the law proposed thirteen papers will be required ; and simplified as much as possible by avoiding the lower officers and applying directly to the judge of a court of record, twelve papers, according to forms prescribed in the bill must be made out before the legal process is accomplished.

The objections to this form of procedure are :

1) It is unnecessarily complicated. It may be carried out in country districts with a fair degree of patience and labor ; but the difficulties of perfecting the process in the larger cities, and particularly in New York and Brooklyn, will be almost insurmountable. We are credibly informed, in fact, that some of our city judges who have seen the bill have expressed their unwillingness to have anything to do with a method involving so much of their valuable time, and will probably refuse to commit patients at all.

2) The position of Examiner in Lunacy will be degraded to a reward for political labors, since the appointment of the two physicians in each case is left to the discretion of the county officials or judge, and not as now to the wishes of the family concerned.

3) A majority of the patients will suffer harm from the visitation of two strange physicians, the visitation of the county official or judge, and the visitation of an officer of the court with a notification of the legal proceedings about to be instituted.

In contradistinction to these facts we have abundant testimony to the effect that simple in comparison as is the existing law of commitment, no person has ever been, through intentional wrong-doing, placed in an asylum as insane in this State, the present method affording ample protection.

But it is possible to make certain improvements in the law now in force, not only in the manner of commitment, but also in the means of regaining subsequent liberty ; and

the suggestions your committee would submit are as follows :

1) There should be no material change in the present mode of commitment by two medical certificates, sworn to, and approved by a judge of a court of record, as provided in the Laws of 1874.

2) Emergency cases should be received for three days without papers of any kind, as specified in the proposed new law.

3) The medical certificates should be more carefully and thoroughly made out, not only in justice to the patient, but also for the benefit of the asylum physicians, who now rarely receive many facts bearing upon the medical history of their patients or upon their mental condition. To this end the form prescribed in the Gallup Bill should be adopted. A few additional questions should be incorporated in the medical certificate for the purpose of determining whether the physicians have informed the patient of their intention of placing him in a hospital for the insane for treatment, in order to guard against the serious harm so often done to patients by removing them to an institution through deception. The State Commission in Lunacy seems to be invested with the power to prescribe the form in which the medical certificate should be made out, and a law regulating this would not appear to be necessary.

4) There should be a section in the law permitting any higher justice, upon application from any patient in an asylum, to appoint at his discretion a commission of two or three physicians to quietly examine said patient as to his mental condition, and upon receiving their report favorable thereto, to discharge him from the custody of the asylum. By the Laws of 1889, chap. 283, § 22, the State Commission in Lunacy is empowered to make such regulations as to the correspondence of patients as would ensure the proper carrying out of this law.

5) A clause should be introduced into the bill providing that nothing in the lunacy laws of the State shall be con-

strued to interfere with the reception and treatment of acute cases of insanity in chartered general hospitals, in the same manner and under the same conditions as patients suffering from other diseases are there received and treated, provided such hospitals have suitable accommodations approved by the State Commission in Lunacy.

(Signed) FREDERICK PETERSON,
Chairman.

C. L. DANA, M.D.,
RALPH L. PARSONS, M.D.,
GEO. W. JACOBY, M.D.,
Ex-officio.

Periscope.

BY E. P. HURD, M. D., LOUISE FISKE-BRYSON, M. D., W. W. SKINNER, M. D., GRACE PECKHAM, M. D., AND ISAAC OTT, M. D.

DELIRIUM TREMENS.

At a recent meeting of the Imperial and Royal Society, of Vienna, (Jan. 10th, 1890), the subject of delirium tremens was discussed.

PROFESSOR MEYNERT regarded this morbid entity as the result of a slow and chronic poisoning of the organism by alcohol. The attack is always preceded by great excesses in the use of spirits or absinthe; it may also be provoked by sudden suppression. It seldom comes on spontaneously, being generally the accompaniment of an intercurrent affliction: an hæmoptysis, a pneumonia, a pleurisy, an epileptic fit, a traumatism; simple mental emotion may serve as an exiting cause. In this respect it resembles the onset of hydrophobia.

Delirium tremens presents a march of extreme regularity. Two distinct periods in its evolution are recognized. In the first, or period of anguish, the patient is a prey to an intense delirium of persecution; unlike other deliriums of the same nature, the danger which threatens the individual suffering from delirium tremens is immediate. The

patient is afraid of being killed by thieves, and to escape the danger which is impending, he will sometimes attempt suicide. This period of anguish lasts about three days; to this succeeds the second period or that of hallucinations and restless dreams. These toxic hallucinations may be of central or peripheral origin, and in the latter case, they are due to the noxious influence of the blood on the peripheral nerves. The patient thinks that he holds in his hands divers objects such as iron, glass, etc. These tactile hallucinations are generalized to the whole surface of the body.

Among the multiple hallucinations of sight, the most frequent is the vision of small objects: mice, rats, beetles, etc. Others see horrible little spectres, hobgoblins, witches glaring at them. Skoda attributed these phenomena to scotomata. It is true that these hallucinations call to mind the scotoma by three definite forms.

It is no less true that the vision of small objects is sometimes wanting, and the patient sees instead of rats and mice, great troops of elephants, or bands of soldiers leaping over a wall and approaching him. Scotomata cannot explain this kind of hallucinations. At the same time, the appreciation of greatness and of volume varies even in the normal state; the patient may then interpret after his own manner the dimensions of images due to scotoma. The hallucinations of delirium tremens are never stable; they are almost always mobile. This phenomena finds its explanation in the fact that the poisoned blood acts continually on the nerve centres, and keeps awake the notions of detail stored up in the cerebrum. The attack ends by a sort of agitated dream and "professional" delirium.

Hallucinations of smell and taste are frequent; the patients complain of perceiving bad odors, or of their food having a detestable taste. The hallucinations of hearing are more marked than those of sight; the patient hears abusive epithets reproaches, obscence propositions, etc.; there are multiple voices, and every word is a menace.

In the discussion which followed, Exner declared that he had been a pupil of Skoda, but did not know that the latter attributed to scotomata the hallucinations of delirium tremens. He (Exner) believed that these phenomena are referable to unequal and abnormal excitations of the retina by vitiated blood.

* * * * *

With regard to the therapeutics of delirium tremens, (which we believe was not touched upon at the meeting aforesaid), it is doubtful if anything has yet been discovered

to supersede the hypnotic and supporting treatment by chloral, opium, capsicum, nutrients, and occasionally, to meet special indications, but only for temporary effect, alcohol. I have been inclined to regard delirium tremens as a form of alcoholic paralysis, analogous to the *tremblement mercurial* of the French pathologists, due to saturation of the nerve cells of the cerebral cortex with the poison, and consequent enfeeblement and perversion of the sensory, perceptive, and motor functions. The tremulousness of the voluntary marches, the tongue, the members; the vain attempts of the inebriate to escape his pursuing spectre, which generally takes on the form of vermin, reptiles, or the most grotesque and unnatural objects; the haggard, distressful expression of the patient, who can obtain no sleep or rest, and whose languishing organic functions testify to the oppression of the nervous and vital forces, all constitute a pathological syndrome which is peculiar to the disease in question. Doubtless the poison should be at once withdrawn, but in some cases a tapering-off method is certainly preferable to a sudden, complete withdrawal, (though such cases are comparatively few), and there are others where a temporary failure of the heart makes the exhibition of a little alcohol imperative. Sleep is undoubtedly the great restorative, and to procure this recourse must be had to some hypnotic. I believe that experience has proved that chloral is much better and safer than opium. The combination of half a grain of morphine and twenty grains of chloral in violent cases is a good one. Chloral will sometimes be borne in very large doses. It is a good thing to associate with capsicum, which is a stimulant to the gastric expansions of the vagi nerves, and indirectly to the cerebro spinal centres, of great and immediate efficacy. Dr. Kinneer, of Portsmouth, England, used to give scruple boluses of powdered capsicum every two or three hours. Patients in less than an hour after swallowing the bolus, would fall into a quiet sleep, from which in three or four hours they would awake "calm, conscious and convalescent."

I have given capsicum in connection with chloral in the form of the tincture, the dose being twenty drops. Its sustaining influence on the nervous centres seems to be marked, and it tends to counteract any depressant effect of chloral on the heart.

According to Dujardin Beaumetz, (see his book "New Medications," translated by me, page 263), paraldehyde is a better hypnotic in delirium tremens than chloral. The

dose is a teaspoonful as often as required to produce sleep.

Urethan has appeared to me too feeble a hypnotic to be of any utility in delirium tremens, and I believe that the same remark is applicable to sulphonal and somnal.

As for nutrients, milk, meat broths, the meat extracts, and other of the protein preparations in the market, are all indicated in such quantities as the generally inflamed stomach of the inebriate will tolerate. Desiccated blood (P., D. & Co., Detroit), is a useful alimentary food product when a powerful and easily assimilated nutrient is demanded.

Lately a new treatment of delirium tremens by large doses of strychnine has been recommended. To Laton, of Rheims, we are indebted for this therapeutic novelty. Laton advised doses of five milligrammes (one twelfth of a grain) by hypodermic injection, or by mouth; these doses to be repeated twice or three times a day. Dujardin Beaumetz has repeated the hospital experiments of Laton with uniformly good success. By experiments on animals, Beaumetz has found "that there exists within certain limits a real antagonism between the action of alcohol and strychnine." Drs. Journet and Bounard also report favorable results from a series of trials in private practice of this remedy in delirium tremens, and in a number of the *Bulletin Général de Thérapeutique* for 1888, appears an article by a Brazilian physician, Ramos, "On the employment of strychnine in delirium tremens," in which he extols the effects of this remedy. He declares strychnine superior to all other remedies, morphine, chloral, paraldehyde, etc., in controlling the disordered manifestations of alcoholism. Strychnine, in his belief, "has in these cases, a substitutive action on the nerve centres, thus antagonizing the excitant action of the alcohol." Ramos would give large doses, hypodermic injections of one-twelfth of a grain, repeated every four or five hours, till the insomnia, agitation and delirium are mitigated or disappear. In some cases, he does not hesitate to push the remedy till as much as a grain is given in the twenty-four hours.

E. P. H.

THE ELECTRICAL RESISTANCE OF THE HUMAN BODY.

("Rivista Sperimentale di Freniatria e di Medicina Legale," vol. xv., fasc. ii.-iii., 1889, p. 226. Observations by Dr. B. Silva and B. Pescarolo.)

The following are the conclusions given by these authors, after a long and able article on this subject:

1. The electrical resistance of the human body to the

galvanic current, great in the beginning, descends at first rapidly and then more slowly, to maintain, after a variable time in different individuals and in the various diseases, a constant minimum for a given electro-motor force. The same facts are observed in the cadaver.

2. The electrical resistance of the human body diminishes with the augmentation of the electro-motor force and of the surface of the electrode, and *vice versa*, and is scarcely influenced by the pressure and the temperature of the electrode, a very little by the temperature of the body, increasing or decreasing with the temperature of it.

3. The interruption has no noteworthy influence on the electrical resistance. Commutation has a greater influence, especially when the surfaces of the two electrodes are very different, since the resistance diminishes, especially at the anode.

4. The electrical resistance of the various parts of the body varies with the thickness of the epidermis, and is in relation with the number of sweat and sebaceous glands. Where these are in great numbers, and where the skin is thicker, the resistance is greater, and *vice versa*. The manner of the behavior of the resistance to the palm of the hand and to the plantar surface of the foot is different from that in other regions; there, in fact, the resistance is great and relatively constant.

5. In fevers, in the exanthemata proper, in correspondence with the greater eruption, in the obese, in the diabetic, in the convalescent from infectious diseases, and in persons with a dry skin,—the electrical resistance is great; on the contrary, it is less, and it advances rapidly to the constant minimum, in the active person with a vigorous cutaneous circulation with much sweating; as also in Basedow's disease.

6. In hysteria the increase of resistance referred to by Vigouroux is not constant; also the difference of the resistance from the two parts of the body in hemiplegia and hemianæsthesia, organic as well as functional, does not always act equally. An influence of the variation of the endocranial pressure on electrical resistance cannot be admitted, and pleuritic effusions make no difference. However, in ascites the resistance of the walls of the abdomen diminishes after the development of the ascitic fluid.

7. With the antipyretics, should the temperature be lowered or not, with sweating or no sweating, in the feverish as in the healthy, we have a diminution of electrical resistance. Bathing diminishes the electrical resistance of the feverish

alone when it diminishes the temperature. It has no evident influence on the healthy. Pilocarpine, whether it produces sweating or not, reduces the electrical tension of the body for the galvanic current. Penciling with oil of mustard has the same effect, as has also application of spray of chloroform, but in a less degree than the former. Venereal abuse, tobacco, emotion, fasting for twenty-four hours, the atmospheric state, abuse of alcohol, atropine, nitrite of amyl, faradization of the skin, have no manifest effect upon the action of electrical resistance.

8. The particular method of behavior of the resistance of the human body after the galvanic current depends upon the modification which it induces in the epidermis, from its anatomical state, which also is influenced besides, as by external agents (traumatic causes, pressure, etc.), by the manner of action of the vaso-motor system, and by the action of various remedies. Hence it is not true that the measure of the electrical resistance serves to represent the state of the vaso-motor system; neither has importance in Basedow's disease, in hysteria, in endocranial affections, etc., being a phenomenon wholly physical and depending on the vaso-motor system only secondarily.

9. The better method of making an electrical diagnosis consists in using the table of Erb, with rheostat in the secondary circuit, the electrode of equal surface (10 cm.), wet freely with water at 40° C., with the fixed electrode in the palm of the hand or in the sole of the foot, proceeding with the greatest rapidity possible. The best electrodes are made of brass or zinc with a layer of mud on its surface held in place by a piece of cloth or leather. They should be applied with a moderate pressure, and possibly always equal, to the skin during the electro-diagnostic examination.

The translator would add that this extremely interesting communication, instituting as it does a line of investigation which should be pushed much farther, should be read *in extenso*. The experimentations and observations, which precede and elaborate the ideas given in the conclusions which have been translated, are worthy of careful attention. G. P.

THE MUSICAL SENSE IN IDIOTS.

The Annales Médico-Psychologiques, January, 1890, contains some interesting references to this subject. Esquirol called attention to the fact that even idiots without

the power of speech could sing. Their musical capacity has recently been tested by Dr. Wildermuth, of Stetten. Children to the number of one hundred and eighty, idiotic in various degrees, have been examined and compared with eighty normal children, in regard to vocal range, the sense of harmony, and memory for melody. Arranging them in four distinct classes, beginning with those who are musical in the highest degree, the following results and proportions have been obtained :

	1st Class.	2d Class.	3d Class.	4th Class.
Idiots. - - - -	27-100	36-100	26-100	11-100
Normal children.-	60-100	26-100	11-100	2-100

This remarkable relative development of the musical sense in idiots is the more striking on account of the utter absence of any other evidence of artistic taste. A beautiful landscape or a lovely picture is powerless to move them. At three months normal children will often manifest great delight at the sound of music, and retain the memory of melodies as early as the first year. The musical sentiment undoubtedly exists among certain of the mammalia (dolphin, seal, mouse, ape [L. F. B.])

The practical outcome of Wildermuth's observations is to impress anew upon the medical mind the necessity, in its training of idiots, of vocal culture, especially the art of singing that is accompanied by rhythmic movements.

In a consideration of "Electricity in Mental Disease," referred to in the same journal, Morel finds that in simple melancholia and in melancholia attonita, electricity brings about the happiest results. In mania, electrotherapy has the narrowest possible field. Hallucinations of hearing are greatly relieved in cases of partial delirium. Dementia and general paresis are not benefitted. In one case out of nine of general paresis not lasting longer than three months, Heyden obtained good results from the use of electricity in regard to various sensations, though the mental state remained the same. Electricity renders important service in functional insanity such as that accompanying epilepsy, hysteria, chorea, and hypochondria.

(Ibid.) Dr. E. Régis has recently called attention to the immense progress made in the care and treatment of the insane, during the past hundred years. While much has been done, there yet remains much to be accomplished. "It is not sufficient," say Régis, of Bordeaux, "to point with pride

to the road already travelled, and applaud the excellence of our predecessors. The world moves, it must move. We have something to do in this matter ourselves, a duty to perform. Everything can be made more perfect ; and however obscure each one of us, we must all work earnestly at a task at once difficult yet full of consolation ; the progressive, constant amelioration of the condition of the insane."

EPILEPTIC ARITHOMANIA.

(Ibid.) Under this title, Dr. A. Cullere, records several cases of epileptics who have a mania for counting, for combining numbers of all kinds, particularly calculations in regard to divisions of time, such as seconds, minutes, hours, days, months, years, and centuries. In one instance, a young man of twenty-seven, and a victim of epilepsy, cried out suddenly to the doctor, in the midst of an attack of acute mania, in which he imagined himself surrounded by brigands and assassins: "Shall I tell you how many minutes there are in one hundred thousand years?" This was an evidence of a temporary return to his abnormal normality, to the individual inherent departure from normal mentality that constituted his natural state. Professional mathematicians and some few children are the only persons who find real pleasure in arithmetical calculation. Possibly the epileptic arithomaniacs resemble such children. With the unfortunates under consideration, this mania for calculations differs materially from similar mania in those of inherited mental instability. The latter are pained and fatigued by arithmetical calculation even to the point of anguish, and of well-defined melancholia. Not so the epileptic. Calculation is an amusement. Even fractions are a source of pleasure. The mysticism of epileptics, their irascibility, their changeable moods, expansive religiousness, sexual aberrations, and alcoholic excesses, are due to inherent mental qualities, as is arithomania, and not to any special hereditary unsoundness of mind.

POST-FEBRILE INSANITY.

In the Johns Hopkins Hospital Reports, No. 1, Vol. ii., seven cases of post-febrile insanity are cited. The prognosis is always good, recovery usually taking place within three months. The patient should be cared for at home whenever possible. Seclusion, incessant watchfulness, absolute rest in bed, with massage and careful feeding, constitute the essentials in treatment.

CHLORALAMID.

According to the *Glasgow Medical Journal*, Dr. Peterson (Lancet, October 26, 1889,) has tested this drug in fourteen cases of insomnia. There were four cases of simple sleeplessness, two being over sixty years of age. The results were most satisfactory, as they were in three cases of phthisis with profuse night sweats. The night sweats were checked in marked degree. In two cases of heart disease, doses of thirty grains gave fair rest, eased pain and relieved cough. Restlessness resulting from pain was but slightly benefited. The undesirable effects were giddiness, feeling of sickness, dryness of the mouth, and even slight delirium. Its action is not so rapid as that of chloral, sleep ensuing only in a half hour or an hour. The doses recommended are from thirty to forty-five grains for a man, and twenty to thirty grains for a woman. The absence of any depressing effect on the circulation makes it an invaluable agent in cases where there is any cardiac affection. This drug is also referred to by Dr. Leech, in the discussion on "Recently Introduced Hypnotics and Analgesias," in the last annual meeting of the British Medical Association. It is adapted to the sleeplessness of nervous people and those suffering from spinal disease, bronchial asthma, subacute rheumatism, and gastric disorders, unassociated with great pain. Dr. W. Hale White (British Medical Journal, December 14, 1889), has given chloralamid in twenty cases in which insomnia was a prominent symptom, with thoroughly satisfactory results with but two exceptions. Some of the patients were suffering from extremely painful diseases, yet the drug produced sleep, sometimes acting better than morphia. Its success was undoubted in cases of enteric fever, malignant disease, aneurism, nephritis, cardiac disease, ascites, erysipelas, rheumatic fever, eczema, phthisis, brachial monoplegia, and spastic paraplegia.

ASTHMA AS A NEUROSES.

The *Medical News* of Jan. 4, 1890, contains a suggestive paper with this title, by J. G. Carpenter. The author thinks that rheumatism and gout play an important rôle in asthma; and during sudden changes of weather or temperature, asthma, bronchitis, or some skin eruption may appear in the absence of a rheumatic or gouty attack. Uræmia, from diseases of the kidneys, may cause the most severe attacks of asthma. That this disease, so much more preva-

lent in childhood, is due, the author thinks, to improper management of the child at birth. Even within a half hour after its advent into this cold world, a rhinitis may be developed from undue exposure and the rapid evaporation from the body and the radiation of heat. The child starts in life with a cold, has continued recurrences, thereby establishing chronic or subacute catarrhal inflammation of the upper air passages, which its sequelæ furnishes the most potent pre-disposition to asthma. Prognosis is good, if proper treatment is given before irreparable structural lesions have taken place. Asthma depends on three conditions: 1. Neurotic habit, as shown by Salter. 2. Diseases of other nasal mucous membrane. 3. Obscure conditions of the atmosphere. To Dr. Loomis the profession is indebted for the use of morphine as an antidote to uræmic poisoning. In complete coma, one-half to one grain injections have been given by him. He claims positive relief of distressing symptoms, and in addition: 1. to arrest muscular spasm by counteracting the effects of the uræmic poison on the nerve centres; 2. to establish free diaphoresis; 3. to facilitate the action of cathartics and diuretics, more especially the diuretic action of digitalis. In renal asthma morphine is a therapeutic remedy of the highest value. The writer believes it has a special effect—also belladonna—on the speno-palatine ganglion.

The speno-palatine ganglion supplies branches to the nose, throat, soft palate, and Eustachian tube. It possesses a sensory, motor, and sympathetic root; and is connected with the pneumo-gastric and facial nerves, and through its numerous connections an intimate sympathetic relation is established between the throat, nose, ear, larynx, trachea, and bronchial tubes. Removal of this ganglion causes a severe catarrhal condition of the nasal mucous membrane. This membrane is continuous with that which lines the eyelids and nasal duct, the throat, Eustachian tubes, the middle ear, larynx, trachea, and bronchial tubes. An irritation or congestion started in the nasal chambers may extend reflexly to the pneumo-gastric nerve, and cause asthma by bronchial spasm; or the irritation may be so great as to cause, in addition to asthma, acute or subacute catarrhal inflammation of the upper air passages and bronchial tubes. Opium and its preparations and belladonna have a specific effect in allaying irritation and checking inflammation and secretion in the upper air-passages by acting on the nerve centres, and are highly important not only during paroxysms of asthma, but in the intervals to assist local treatment in allaying chronic irritations and congestions. In

connection with quinine and nux vomica, they exert a powerful tonic influence on the vaso-motor nervous system. In tonic doses thrice daily, they prevent the return of asthma while the intra-nasal disease is being cured locally. Nitroglycerine has an important place during the paroxysm and in the intervals of respite. Chloral hydrate allays the attacks. When the paroxysms are violent and threaten life, chloroform is of great use.

L. F. B.

DIPSOMANIA.

T. S. Clouston, M.D., F.R.C.P.E., in the "Edinburgh Medical Journal," for February, 1890, continues his communications on the subject of "Diseased Cravings and Paralyzed Control," by treating of dipsomania, which he attempts to define. He first refuses to apply that name to those forms of mental disease in which an intense craving for alcoholic drink is merely a prominent symptom, and deprecates the careless use of that term by practitioners who apply it to such states and employ it inaccurately. Thus cases of simple coherent mania—that is, with distinct mental exaltation, insomnia, restlessness, talkativeness, changed habits, loss of common sense, morbid brilliancy of imagination, and hyperæsthetic memory—must not be called cases of dipsomania, though such patients may drink excessively, have all their symptoms aggravated by it, and have an intense craving to get it. He also cited a case of *folie circulaire*, in which the phase of exaltation always began after abuse of ardent spirits, which he craved and obtained at all hazards as long as it was possible to do so; yet this was not true dipsomania.

In simple melancholia, in epilepsy, in many cases of mild dementia, there are often manifested quite uncontrollable longings for drink, as also in some cases of delusional insanity, paranoia, and general paresis, and even in rarer cases of softening of the brain, tumors, cerebral syphilis, in which a craving for alcohol was one of the earlier symptoms. Drink-craving with loss of control is sometimes one of the early signs of the break-down of senility. But a dipsomaniac, while he remains a pure case of that disease, has no systematized delusions, no amnesia, and no motor symptoms, and has seldom strong suicidal or homicidal impulses. The greatest difficulty in the diagnosis of dipsomania is to distinguish it from drunkenness, in which, however, the control is not paralyzed, but simply not exercised; whereas, in true dipsomania, the power of control is abolished. It is therefore a form of diseased craving or impulse, with paralyzed—wholly or partially—inhibition, and may be divided into four classes :

1. *Developmental and retrogressive dipsomania*, which includes the congenital cases, whose higher inhibition had never been developed as a brain-faculty.

2. *Dipsomania of a neurotic diathesis*, comprising those cases having high brain qualities, or keen sensibilities and poetic minds, or of hyperæsthetic conscientiousness, but who are carried away by the force and intensity of their emotions, and lose control over their cravings.

3. *Somatic dipsomania*, or cases in which traumatism, sunstroke, paralysis, cephalic erysipelas, cerebral lesions of all sorts, so weaken the self-control that men, who had previously led sober lives, then acquire marked and uncontrollable cravings for liquors.

4. *Dipsomania of excess* includes those forms in which there is no especial heredity, no neurotic diathesis, no disease, and no critical period of life, and where there has previously been a prolonged and excessive use of stimulants.

In regard to treatment, he recommends "legal control" for many cases—*i. e.*, enforced abstinence in an asylum, total abstinence in free individuals, special asylums for those willing to be treated there, the employment of every means to strengthen the bodily health, the judicious use of special expedients (drugs), and, as a means of prophylaxis, the correction and development of the weak points in the children of the first division.

TREATMENT OF DIPSOMANIA BY HYPNOTISM.

Dr. Hayes, secretary of the London Hypnotic Society, comes forward with another statement in favor of the employment of hypnotism in dipsomania. The previous publications on the subject by Ladame, Forel, and others are also in support of the belief that long periods of abstinence and even permanent cures are obtainable by this method. While the idea of curing dipsomania by hypnotism and suggestion is not a very new one, yet it is well to call the attention of the medical profession to it, in order that it may be more widely known and practised. The patient submits to hypnotism two or three times a week, and, when he is in the proper condition, the hypnotist suggests to his now pliant and receptive mind that he will have no desire for drink on awaking and that he will even have a repugnance for it. After several *séances* of this nature the desire diminishes, ceases altogether, and the patient loses the habit of drinking alcoholic beverages. Relapses often occur, it is true, but, on the other hand, cures are often permanent.—*Quarterly Journal of Inebriety*, January, 1890.

EXPERIMENTS RELATING TO THE ACTION OF ALCOHOL
ON THE BRAIN.

Dr. J. J. Ridge, physician to the London Temperance Hospital, publishes the results of his experiments in the "Medical Temperance Journal." He admits that alcohol acts as a narcotic, thereby dulling the action of the cortical centres. His experiments, which required a considerable degree of mental as well as muscular alertness, consisted in the endeavor "to pass a pointed stick through a swinging ring, counting the number of swings between each successful endeavor, and adding these together when sixty had been accomplished. A certain dose of rectified spirit was then taken, and after fifteen minutes the number of swings required to accomplish sixty more successes was counted."

The following table shows the results of his experiments :

<i>Alcohol.</i>	<i>No. of Swings before.</i>	<i>No. of Swings after.</i>	<i>Percentage of Increase.</i>
1 drachm	153	169	10.5
	113	126	11.6
	112	123	9.8
2 drachms	166	194	16.9
	145	156	7.5
	132	154	16.6
	125	146	16.8
3 drachms	134	185	38.0
	115	142	23.5
4 drachms	141	204	44.6

It is thus seen that, after the ingestion of the alcohol, he was less successful in passing the stick through the ring than before taking that agent, and it is also seen that the degree of insuccess was approximately proportional to the quantity of alcohol absorbed. Each experiment was done on a different day, so that no influence upon the results can be attributed to fatigue. The several functions of the brain and spinal cord that are tested in these experiments are: 1st, the steadiness of the hand (co-ordination of muscles); 2d, visual acuity; 3d, accuracy of judgment; 4th, rapidity of thought (perception and decision); 5th, rapidity of muscular action; 6th, power of self-control.

The experiments of Kraepelin, published by Lander Brunton, in his text-book of pharmacology, point in the same direction. This experimenter studied the influence of

alcohol in three forms of tests, viz.: (a) to find the time required for simple reaction, *i. e.*, for a message received by the senses and returned by the motor nerves; (b) for discrimination; (c) for decision. In all of these tests, involving a certain interposition of cortical activity, the mental processes were found to be prolonged and retarded by the absorption of alcohol by the person under experiment.

DRUNKENNESS: ITS INFLUENCE UPON THE MIND.

Dr. T. L. Wright, of Bellefontaine, Ohio, has an article in the "Quarterly Journal of Inebriety," for January, in which he studies the effects of alcohol upon the mind. He rightly disparages the common opinion that this substance really adds to the power, scope, and brilliancy of intellectual operations, and truly remarks that the poison impresses and modifies the mental faculties separately and in detail, and also throws an undefined and immovable glamour over the mind as a whole, so that it is quite incapable of correctly judging of its own condition. The *attention* of an intoxicated man is more difficult to enlist than that of a sober man, because, his nervous sensibilities being enfeebled, he is not thoroughly alive to ordinary sensations and impressions, and for this reason his ideas become fixed, his opinions unchangeable, while in this state. He may not even rightly feel the flight of time, and be astonished when told how late it is. Under the same circumstances wonderful *egotism* becomes developed in him. The geniality and good-nature of new intoxication are intensely selfish. He loses his affection, love, or regard for others, and manifests a morbid feeling of grandeur united with one of condescension for others. When used as a stimulant by public speakers, alcohol is apt to cause a superficial fluency of speech, while really detracting from its merit or wit. It causes the expression of empty assumptions and baseless exaggerations, and the delivery of a string of pompous or high-sounding verbiage, instead of words pregnant with thought. It also produces a diminution of sensations and a dullness of perception, and often an aberration of the latter, so that illusions, hallucinations, and delusions are produced. The idea that somehow *he is physically invulnerable* is no doubt largely due to the dullness of sensation and perception in the person intoxicated, and this idea is no doubt the cause of those rash exhibitions of reckless courage often given by intoxicated persons.

The influence of alcohol upon *consciousness* is well known. All the mental processes included under that term are impaired by it. Memory, judgment, discrimination, are para-

lyzed by alcoholic anæsthesia. Reasoning is impossible, for the "data of consciousness" are dim, imperfect, or absent.

The author concludes by observing that alcohol in small quantities will render consciousness dim, feeble, unreliable, while in larger portions it will disorganize the powers of consciousness, or will totally wreck and destroy it.

ON THE DIAGNOSIS OF NEURALGIA IN AND ABOUT THE EYES.

Dr. A. D. Williams calls attention to a symptom attending neuralgia of the fifth pair, which he justly considers quite important, viz.: the tenderness of the skin after the attack has passed off, or even while the pain is present. Touching of the scalp or slight pulling of the hair is painful. The eyeball may also be tender to the touch. Other aids to diagnosis are the frequent periodicity of neuralgia, and the fact that if any considerable inflammation is present the pain is *probably* not neuralgic.—*St. Louis Med. and Surg. Journal*, Feb., 1890, p. 109.

IODOFORM IN CEREBRO-SPINAL MENINGITIS.

"In the Tchernigov weekly 'Zemsky Vratch,' No. 10, 1889, p. 151, Dr. G. Levitsky, of Vostrovskaja, calls attention to excellent effects in cerebro-spinal meningitis obtained from the internal administration of iodoform given in the form of two-grain pills, three times a day. He reports a striking case, that of a woman suffering with an exceedingly severe form of the disease, in which, after all other means had utterly failed, the administration of the drug was almost immediately followed by a steady improvement. On the third day of the treatment contractures of the right, and on the fifth of the left, upper limb disappeared; by the end of the fourth week the patient was practically well. The drug was therefore discontinued. A relapse, however, rapidly followed, but yielded at once to another course of iodoform; a complete and permanent recovery taking place ultimately. In all, *one ounce* of iodoform was taken in the course of two months. No untoward accessory effects were ever observed."—*Canada Med. Record*, Jan., 1890.

THE BROMIDES IN EPILEPSY.

"Dr. Moritz Gauster, whose extensive experience in the treatment of this disease enables him to speak authoritatively, concludes as follows: (1) The bromide treatment in

epilepsy is the most successful, particularly in idiopathic cases. (2) As a rule, the bromides must be administered for years, the dose in each individual case being regulated by observation. (3) By careful observation of the condition of patients, as much as 20 grammes can be given daily without manifest injury. (4) The bromides must be suspended or supplanted by other agents. (a) When digestive disturbances supervene; when slight they are of no consequence, and generally disappear, notwithstanding their continued use; (b) when catarrh of the pulmonary apices can be detected; (c) when ulceration of the skin or any cutaneous complication exists. (5) Involvement of the intelligence does not indicate a discontinuance of the bromides. (6) Pulmonary tuberculosis, severe cutaneous lesions and grave nutritive disturbances alone forbid the bromide therapy. When combating the attacks of epilepsy this is not of such vital importance as preventing the supervention of severe psychoses. (7) Emaciation is no contra-indication, as the weight may increase when sufficient nutritive elements are ingested. (8) During the treatment attention must be directed to the nutrition, and at intervals to the lungs and skin.—*Wiener Medizinische Presse*. From *Canada Med. Record*, Jan., 1890.

THE DISPOSITION TO BE MADE OF CRIMINAL LUNATICS.

Dr. W. W. Godding, superintendent of the Government Hospital for the Insane at Washington, discusses the three methods of disposing of insane criminals, viz.: Retention in an asylum, hanging, and release. The last method can not, of course, be put into practice, as the safety of the public requires that the homicidal maniac be deprived of his liberty. The second method is certainly sure and radical, admitting of no revision, and freeing the community from a dangerous individual. This method of treatment by hemp is evidently the simplest of all, yet it does not accord with our present views of sociology. The author naturally concludes in favor of the first-named method, and also adds another plea for the rational and humane treatment of insane criminals, which consists in their removal from the ordinary hospital for the insane to a special department of an asylum, or State hospital for the insane, where they will be cared for in a suitable manner. He closes with a description of Howard Hall, a building attached to the Government Hospital at Washington, in which the criminal insane are thus disposed of.—*Medico-Legal Journal*, Dec., 1889.

SYMPTOMS OF GRAVE DISEASES OF THE NERVOUS SYSTEM
RELIEVED BY RESTORATION OF THE EQUILIBRIUM
OF THE MUSCLES OF THE EYE.

Dr. J. F. Fulton, professor of ophthalmology and otology in the University of Minnesota, recounts his successes in this direction. In 260 cases of heterophoria in which he operated, cephalalgia was present in 190 cases. In the vast majority of these cases the headache was cured; in nearly all relieved. This fair degree of success prompts him to go to the extreme and to say, that before *any* case of headache is treated, the condition of the ocular muscles should first be tested. In three cases which he terms chorea of the lids and face in children between ten and twelve years of age, the symptoms were completely relieved by a partial tenotomy of some one of the recti with correction of the abnormal refraction. He has had no experience with epilepsy. Neurasthenia in a business man of forty, who had vainly consulted the celebrities in neurology of the metropolis of four countries in both hemispheres, yielded to his magic touch. Even cases of hemianæsthesia, with partial unconsciousness, vertigo, complete aphasia, diplopia, and hemiopia, causing the physician in charge to fear the existence of some cerebral lesion, were cured by a few snips of the scissors, of a stroke or two of the knife. In closing, however, he says: My conclusions are exactly those of Dr. Webster, viz.: (1) No person should have a tenotomy performed *solely* because he is the subject of heterophoria. (2) But slight degrees should be corrected where troublesome symptoms exist which may be due to the too great use of nervous force in co-ordinating the eyes. (3) Other means should be resorted to before trying tenotomy, but unnecessary delays should be avoided. (4) Tenotomies should be performed under cocaine. (5) In judiciously selected cases, where the operation is properly performed, the average results will be quite as satisfactory as the results of most other surgical operations.—*Northwestern Lancet*, Feb. 1, 1890. W. W. S.

THE EFFECT OF STIMULATION ON THE POLARIZATION
OF NERVE.

Dr. George Stewart has made an elaborate study of the subject. In his first paper he determines whether stimulation has any effect on the polarization of a nerve. Hermann assumed for the explanation of the apparent change of resistance produced in a nerve by tetanizing that the positive polarization during the flow of the polarizing current

is increased by stimulation. The question then is, how is the polarization after current affected by stimulation? He studied this in two ways. In one the polarization was allowed to become pretty steady and then the nerve tetanized and the effect observed. In the other the stimulation was continued during the whole flow of the polarizing current and the amount of polarization compared with that produced by a similar current when the nerve was at rest. These two methods were used, but as a matter of fact the latter method is not suitable for long periods of flow as the former, because the prolonged tetanus exhausts the nerve. There is this difference between the methods, that in the first case the stimulus acts upon a polarization already established, while in the second it may be supposed to influence the establishment of that of polarization. His apparatus was arranged so that any one of the three circuits could be closed at will. In one of these the circuit was the nerve and battery, in the second the nerve and the galvanometer, and in the third the nerve battery and galvanometer. The sciatic nerve of a frog was used, and his results were as follows :

1. The effect produced by stimulation is in the direction of diminution of the positive polarization. 2. Within limits the effect is somewhat greater the longer the time of flow of the polarizing stream. 3. The effect increases within limits with the density of the polarizing current. 4. As might be expected the effect increases with the strength of the stimulus. In his second paper he considers more in detail the work of his preceding paper. The object of the inquiry was to investigate the electrical changes which take place in a polarized nerve when it is stimulated both during the flow and after the opening of the polarizing current, so far as these changes have not hitherto been studied or where the study of them has been incomplete. He tried to obtain not only qualitative but also quantitative results, relying in the latter case rather on comparison of different experiments upon the same nerve than on observations made on different nerves at different times.

1. Galvanometric observations during the flow of the polarizing current where the whole intra-polar area is led off to the galvanometer—I. Stimulating electrodes in extra-polar region—here with stimulation on the anodic side, a limiting intensity of current can be reached for which the stimulating effect disappears altogether.

2. Stimulating electrodes in intra-polar region. When we come to the comparison of the extra and intra polar effect with intra-polar stimulation, we shall find additional

evidence to show that the anode becomes impassable about the time when the intra-polar area in general has lost its conductivity and excitability. On the other hand, it is seen that long after the cathodic block has developed itself intra polar stimulation is effective, producing indeed something like its maximum effect with the current density which corresponds to the beginning of the block.

II. Galvanometric observations during the flow of the polarizing current, when an extra polar area is led off to the galvanometer. 1. Stimulating electrodes extra polar. Here it may be said that in general even 20 after opening the polarizing current the after effect on the cathodic side is greater than the effect during the flow, while on the side of the anode this relation is reversed. 2. Stimulating electrodes in the intra polar region. Here the chief points of difference between these results and those with extra polar stimulation are, (1), the almost complete absence of any stimulation effect on the side of the cathode, except with the weakest currents, and (2), the absence of the positive effect on the anodic side.

III. Observations during the flow of the polarizing current, when the whole intra-polar area is led off to the galvanometer alternately with an extra-polar area. Here the intra-polar effect and the extra-polar anodic effect rise and fall together. The maxima and minima of the two effects correspond closely enough. From the preceding data he arrives at the conclusion that the stimulative effects during the flow of the polarizing current cannot be explained entirely as secondary action currents, but are probably due to the super-position of such currents on electro-tonic variations.

IV. Observations after opening the polarizing current when the whole intra-polar area is led off to the galvanometer. 1. Stimulating electrodes, extra-polar. Here the stimulative effect may fail altogether after the opening of a strong current if the excitation has to pass the anode, while if it has only to pass the cathode we may have a large effect. 2. Stimulating electrodes, intra-polar. Here the direction of the polarizing current is without influence on the amount of the stimulation deflection.

V. Observations after opening the polarizing current where an extra polar region is led off to the galvanometer. 1. Stimulating electrodes, extra polar. When the polarizing current is ascending we get a cathodic after current in the same direction. When the polarizing current is descending, the main anodic after-current in the opposite direction is the one which affects the galvanometer. 2. Stimulating

electrodes, intra-polar. Here as before the sign of the effect is positive in the anodic area, negative in the cathodic.

VI. Observations after opening the polarizing current when the intra and extra polar regions are led off alternately to the galvanometer, the stimulating electrodes being extra polar. The general result was that a large extra polar effect was associated with a small intra-polar effect.

VII. Observations after opening the polarizing current when a part of the intra-polar area is led off to the galvanometer.

(1.) Stimulating electrodes, extra-polar. When the excitation has to pass the cathode the effect diminishes, when any considerable portion of the intra-polar area lies between it and the led off part and it disappears ultimately.

(2.) Stimulating electrodes, intra-polar. Here in general, when the anode is in the led off area, the effect is the greatest.

VIII. Observations after opening the polarizing current when the whole intra-polar region and part of it or two different parts of it are led off successively to the galvanometer, the stimulating electrodes being extra-polar. The results are that except with the strongest currents the effect is greater when the galvanometer is connected with the anode than when it is connected with the cathode.

IX. Experiments on muscular contraction, with intra-polar stimulation.

(1.) During the flow of the polarizing current.

Up to a certain strength of current a stimulus will give contraction when the cathode lies next to the muscle, which will give no contraction when the anode is in that position. Above this strength the reverse holds good and a stimulus which is followed by contraction when the excitation has to pass the anode, evokes no response when it has to pass the cathode.

(2.) After opening the polarizing current his results were as follows :

1. With a very short time of closure. *a.*—If we adjust the stimulus so that contraction shall occur immediately on opening the descending current there will be no contraction for some time after opening the ascending current. *b.*—If we adjust the stimulus so that contraction shall not occur for some time after opening the descending current it will take a longer time to appear after the ascending current.

2. If we determine the strength of the stimulus which is just necessary to give contraction immediately after opening, it will come out greater for the ascending than for the descending current.

3. When an interval has existed during which the stimulus has been inoperative, the contraction does not reach its original height for some time after the stimulus has again become effective, and this time is longer after the ascending than after the descending current. He does not see how the conclusion can be avoided that after opening the polarizing current a rapid, perhaps instantaneous reversal of the relations of the poles takes place.

The remainder of the paper is devoted to a discussion of preceding facts in regard to conductivity and excitability. He states, "I do not now say that excitability and conductivity are separable properties. What I say is that we know nothing in the manner of propagation of a nerve impulse which goes against such a supposition. We know much which supports it."—*The Journal of Physiology*, vol. ix., No. 1, 1888; and vol. x., No. 6, 1889. ISAAC OTT.

EXCERPTS FROM ITALIAN JOURNALS.

BY ALBERT PICK AND F. H. PRITCHARD, M.D., BOSTON.

ON TWO CASES OF RUMINATION IN THE INSANE.

Dr. G. B. Verga (*Archivo Italiano per le malattie nervose*, 1886, xxvi., p. 149). The writer, after giving a review of the relatively rich literature on rumination in man, communicating at the same time two cases of his own with post-mortem results, turns to the explanation of this phenomenon. That it is not a disease which most writers admit is seen from the most various pathological results found in patients with this symptom, as well as a perfectly normal condition of the organs of deglutition and digestion; especially does he object to those writers, who, like Cantanaro, would have it regarded as an atavistic sign. Even Cantanaro had to confess that very many of his 69 cases from literature, of which only 36 were mentally abnormal, had not ruminated from childhood; hence it was to be regarded as an acquired peculiarity—according to the writer, as a bad habit. Thus it is chiefly found in voracious individuals, for example, in imbeciles, then in people who have no time to eat, and especially in children, in business men, learned men, etc., and finally in persons with faulty apparatus of mastication.

Hence, one should strike merycismus from the list of degenerative and atavistic phenomena.

SURGICAL OPERATIONS AS A CAUSE OF INSANITY.

Dr. R. Gucci (*Rivista sperimentale di Freniatria*, 1889, xv., p. 50). The writer, in his excellent treatise on surgical operations as a cause of mental disturbances, first excludes

all those cases where there was a cerebral concussion as a cause of origin of psychoses, and from his careful study of the literature and from observations :

1. Ovariectomy in a seventy-four-year woman, with mania during convalescence, three months after the operation.

2. Ovariectomy in a thirty-one-year-old woman, with attacks of violent excitement ten days after operation, which returned and led to imbecility.

3. Ovariectomy in a forty-two-year-old woman, who had an insane attack in the twenty-fourth year, and then for sixteen years remained completely well, and only four weeks after the operation was she attacked anew.

4. Enucleation of the eyeball in a sixty-year-old woman, with immediately following grave melancholia.

He comes to the conclusion that in rare and exceptional cases an operation may be the cause of origin of a psychosis. Contrary to the rarity of its appearance, he does not think it a contra-indication in individuals predisposed for an operation which otherwise would have to be performed.

Psychoses seems most frequently to follow operations on organs which have an intimate nervous connection with the central nervous system, as the sexual organs and those of sense.

Further, the fever, the loss of blood, and especially the chloroform narcosis, may have a certain influence.

CHOREA HEREDITARIA (HUNTINGTON'S CHOREA).

CHRONIC PROGRESSIVE CHOREA.

Dr. G. Leppilli (*Revist. sperimental. di Freniatr. etc.*, 1888, xiii., p. 453) gives a record of the cases of the disease known up to date, and forms a clinical picture of this rare disease, essentially distinguishing itself by its constant transmission by heredity, often through many generations and then making an outbreak later on, generally between the thirtieth and fortieth years, and by its gradually progressive functional disturbance of always new groups of muscles (even of the tongue), and by its incurability from the usual chorea. The abnormal movements are aggravated by emotions, while they, contrary to the usual form of chorea, may be more or less completely suppressed by intended motions; during sleep they cease completely. Disturbance of sensibility are wanting; the mechanical and electric irritability is unchanged, the reflexes are normal, exceptionally heightened. Especially after a more or less duration of the disease are slight psychic disturbances

strikingly frequent, which may even increase to melancholia with inclination to suicide. This disease, dependent upon a hereditary neuropsychopathic base, is not rarely accompanied by a general progressive dullness of the mind. The members of a family afflicted with this form of chorea who remain free are also frequently demented, or at least bazarre and "nervous." If one generation has been spared, then their posterity may be regarded as immuned.

A MODIFICATION OF WEIGERT'S METHOD FOR THE COLORATION OF NERVOUS CENTRES.

G. Vassale (*Revista speriment. di Freniatr. ed. medicina leg.*, etc., 1889, xv., p. 102) recommends the following modification of Weigert's method of coloring by hæmatoxyline, where the objects are hardened in Müller's solution or in a kali bichromate solution and kept in alcohol. He uses three solutions.

- 1) Hæmatoxyline, 1 gr. in 100 gr. of hot distilled water.
- 2) A saturated and filtrated solution of cupric acetate.
- 3) Borax, 2 gr. and kalibichromate, 2.5 gr. in 300 gr. of distilled water.

The sections are first placed into solution 1 for three to five minutes, then the same time in solution 2, and after a rapid cleaning are thrown into solution 3, where they lose their color again. Then they are washed again and the water removed by absolute alcohol, brightened up in a carbo-xylol solution (1 carbolic acid, 3 xylol), and then laid into xylol—Canada balsam. After the color being taken out in solution (3), they may again be colored in alum, carmine or picrocarmine.

THE EFFECTS OF EXTIRPATION OF THE CÆLIAC PLEXUS.

A. Lustig (Turin) (*Archiv. per le Scienze med.*, 1889, xiii., 6). This experimenter used dogs, and especially rabbits. The peritonæum was opened and the plexus removed; the technique may be read in the original. Eleven successful rabbit and two dog-experiments yielded the following: The gastro-intestinal tract showed no disturbances. A few hours after the operation a transient glycosuria appeared, which at the most continued two days. An atrophy of the pancreas never appeared. In the first days—sometimes with and sometimes without mellituria—*acetonuria* appeared. This often lasted until death, with continued decrease in the body-weight and the temperature, and with slowing of the respiration; death generally appears in a few weeks from coma aceticum. Some of the animals recovered. The changes in the kidneys were those of acetonuria.

P. & P.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY

Stated Meeting, February 4, 1890.

PRESIDENT, DR. GEO. W. JACOBY, in the Chair.

Dr. CHRISTIAN A. HERTER described a case of
MULTIPLE CEREBRAL SOFTENING, WITH WIDESPREAD EN-
DARTERITIS AND A DISSECTING ANEURISM OF A
BRANCH OF THE LEFT MIDDLE CEREBRAL
ARTERY. (See page 156)

DR. FISHER said that he had observed many cases of endarteritis and softening among the aged in the city almshouse. He described the case of a woman who was dull and stupid, had had two attacks of aphasia, together with hemiplegia, and who was syphilitic, syphilis and alcoholism being common to most of the almshouse inmates. In this patient there were undoubted occlusion of arteries and softening. He believed it would be impossible to distinguish between syphilitic and atheromatous disease of the vessels. He had noted as quite characteristic of specific cerebral endarteritis extreme dullness and stupidity. Often the pupils were irregular and sluggish in their reaction to light. He would consider the hebetude in Dr. Herter's case as due to the general condition of the vessels rather than to the lesion in the frontal lobe.

Dr. STARR asked the reason of the asymmetry of the hemispheres presented in the drawings.

Dr. HERTER said that œdema of the left side was the cause of the disproportion. In reply to Dr. Fisher he stated that his case was not syphilitic and the age was but 32. Naturally softening was common in people of advanced age.

Dr. FRANK H. INGRAM then read a paper entitled
A CONTRIBUTION TO THE STUDY OF EPILEPSY. (See page 165)

Dr. STARR thought it interesting to hear of the apparent relation existing between changes of barometric pressure and the frequency of epileptic attacks. There was nothing which tended to produce epileptic seizures so soon as variations in arterial tone, a fact which might possibly serve to explain the matter of atmospheric influence.

As regarded the pathology of epilepsy there seemed to be no permanent lesion of the brain; and all such gross changes as had been described by Alexander, by Meynert and others, were not the direct cause of the disease. It was inconsistent with our clinical knowledge of the disorder to seek for visible pathological changes.

The treatment of epilepsy differed much in its results in dispensary and private practice, the former cases existing under such bad hygienic conditions. Contrasting the two classes, dispensary cases had six times as many seizures and were in other ways worse than private patients. There was much of value in the character of the aura. Dr. Ingram reported no aura in 50 per cent. of his cases. Undoubtedly the point of departure in epilepsy was critical, and the character of the aura gave the seat of the discharge. If the aura were visual, as in many cases, the point of origin was in the visual area. If auditory, a rarer phenomenon, it began in the auditory area. Although the larger proportion of epileptic seizures was due to cortical disturbances, such discharge might take place from gray matter anywhere in the nervous system. It was unfortunate to condemn the bromides, for although often injurious, they gave better results than any other known drugs, when employed under proper regulations.

Dr. HERTER agreed with Dr. Starr that there was no relation between the pathological findings in epilepsy and the disease; but nutritive changes in an unstable cortex were probably the cause, apart from any gross pathological lesions.

Dr. FISHER thought that the bromides did not interfere much with bodily nutrition, as many patients grew fat on them. They seemed to become habituated to them.

Dr. SKINNER described a case where the cutting off of the bromides resulted fatally. The patient, a young woman, had been for some time under bromides, when she was sent to an oculist to have her eyes examined. The latter found mixed astigmatism. The bromides were cut off. After three weeks she began to have *petit mal* very frequently; they became more and more frequent, until finally she sank into coma and died. One and one-third grains of morphia in four doses hypodermatically made no impression upon the seizures. The fits invariably began upon the right side of the body, with deviation of the head to the left and of the eyes to the right. He thought there had been a cortical hemorrhage.

Dr. LESZYNSKY said that the autopsy in cases of status epilepticus yielded no result. Patients died from heart or respiratory failure. He believed he had saved the lives of several such cases by venesection. He had used nitrite of amyl before he knew that it was harmful. Most drugs are of no service, with the exception of chloral, which in 40-50 grain doses per rectum had acted well. The indiscriminate use of bromides in epilepsy was injurious, but their careful administration was productive of satisfactory results.

Dr. LYON had employed pilocarpine in a case with epileptic convulsions, and with excellent results. It produced

first a profuse perspiration, after which the patient emerged from his attack. In asylums it was very common to withdraw the bromides from cases, but he had never observed any harm to follow. He had used pilocarpine also successfully in a case of hystero-epilepsy.

Dr. HERTER thought pilocarpine should always be employed with the greatest caution. He had seen it produce pulmonary œdema and death in two cases.

The PRESIDENT related the case of a barber who several years ago began to fall asleep when at his work, and was consequently discharged. The somnolent attacks had continued. He would fall asleep while walking, or riding on the platform of cars, and had frequent falls into the street, into gutters, on the stove, etc., none of these things waking him up. There was no convulsion, nothing to call epileptic. Ten years ago he weighed 150 lbs., now he weighs 270 lbs. Curiously enough he was a sufferer from insomnia, not being able to sleep continuously at night for more than half an hour. Were these epileptic attacks? Was there any connection between them and the corpulence?

Dr. DANA had reported a case of epileptic morbid somnolence in a young woman several years ago. She had had at first only somnolent attacks, later developing real epilepsy. He believed these somnolent seizures to be a form of *petit mal*. He had had a case similar to Dr. Jacoby's in conjunction with Dr. Hammond. He walked about while asleep, but did not hurt himself, and could be roused. The pupils were contracted as in normal sleep, and not dilated as in epilepsy. It might be allied to narcolepsy.

Dr. INGRAM said that his routine treatment of the status epilepticus had been 60 grains of chloral per rectum every two hours, and this had been very successful in the majority of cases. He had also seen good results and no injury from the use of pilocarpine.

The Committee, consisting of Drs. Peterson, Dana and Parsons, appointed at the January meeting to examine the

GALLUP LUNACY BILL,

then made its report. (See Report, page 181).

Dr. STARR expressed himself as thoroughly in sympathy with the criticisms and recommendations of the report. He moved that the report be adopted and printed and that copies be forwarded to the Governor of the State, the State Commissioners in Lunacy, and to such others as were interested in the insanity laws.

After some further favorable discussion by Drs. Fisher, Leszynsky and Ingram, the report was adopted as read, and the recommendations of Dr. Starr ordered.

New Instruments.

A TEST OF WILSON'S CYRTOMETER.

By H. J. MULFORD, M.D.

Extract from a Thesis, to which honorable mention was accorded, presented to the faculty of the medical department of the University of Buffalo in Jan., 1889.

The cyrtometer was designed for the purpose of locating the position of the fissure of Rolando on the living head. It is described as follows:

"The instrument consists of three strips of flexible metal and a taper (D, Fig. 1) for securing it *in situ* (Fig. 1). The

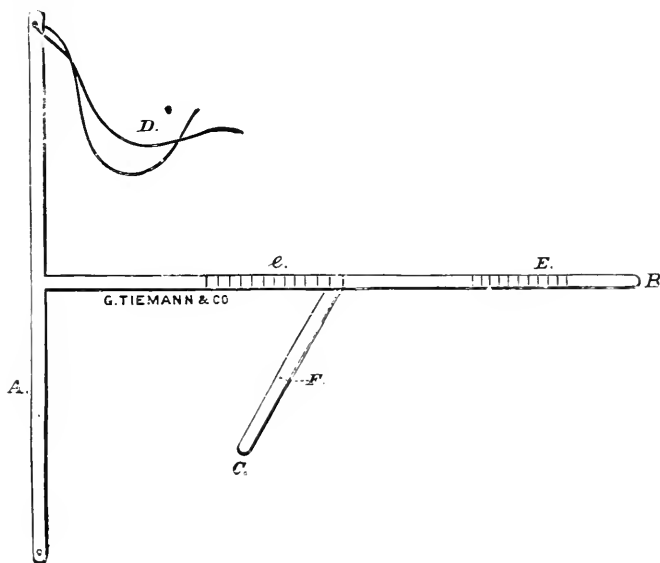


FIG. 1.—The Cyrtometer.

broadest transverse strip (A, Figs. 1 and 2) passes coronally round the forehead, corresponding with the glabella and the external angular process (C and e a p, Fig. 2); the narrower longitudinal strip (B, Figs. 1 and 2) passes backwards from the glabella in the middle line to the occiput. This strip is marked with two scales of letters: capitals in its posterior fourth, and small letters about the middle of the strip (E, e, Fig. 1).

"Measured from the glabella backwards, the distance to any given small letter is 55.7 per cent. of the distance from the glabella to the corresponding capital letter; thus, when

any capital letter falls directly over the inion, the corresponding small letter will coincide with the top of the fissure. A third narrow reversible strip (C, Figs. 1 and 2) slides on the longitudinal slip, making an angle of 67° , open-

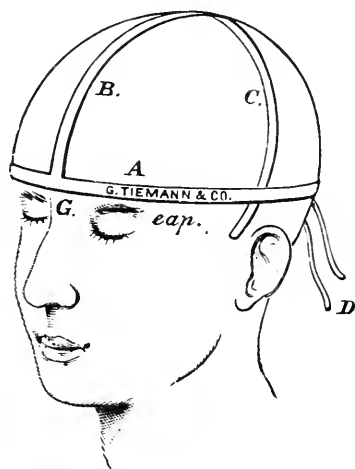


FIG. 2.—The Cyrtometer in position.

ing forwards, and marked at $3\frac{3}{4}$ inches from its attached end (F, Fig. 1), thus giving the length and direction of the fissure on the surface of the head."¹

The following are the scales :

CAPITALS.					SMALL LETTERS.				
<i>Inches from glabella.</i>					<i>Inches from glabella.</i>				
A	(11.5)	-	-	-	a	(6.4)			
B	(12)	-	-	-	b	(6.6)			
C	(12.5)	-	-	-	c	(6.9)			
D	(13)	-	-	-	d	(7.2)			
E	(13.5)	-	-	-	e	(7.5)			
F	(14)	-	-	-	f	(7.7)			
G	(14.5)	-	-	-	g	(8)			

The cyrtometer used by me differed a little from the above. It was made of elastic metal ribbon, such as is used for clock-springs, without a tape, its own elasticity being enough to keep it in position. For this idea I am indebted to Dr. Roswell Park, who was then, and is now, doing considerable surgery of the brain.²

¹ Hare, *Lancet*, 1889.

² Park, pamphlet, "Surgery of the Brain, based on the Principles of Cerebral Localization."

I applied the cyrtometer to eight heads. The following programme being carried out in each case:

First. The hair was removed from scalp.

Second. Cyrtometer was applied over bare scalp. The indicated position of fissure was then marked on brain by pieces of wood thrust through drill hole in skull wall.

Third. Scalp and calvarium removed. Brain examined to see how near indicated position of fissure approached fissure. Length of fissure taken.

TABLE OF MEASUREMENTS.

<i>Case.</i>	<i>Sex.</i>	<i>Distance from glabella toinion.</i>	<i>Distance from glabella to top of fissure.</i>	<i>Location.</i>	<i>Length of fissure.</i>
1.....	Female.	12.25 in.	6.8 in.	Over fissure. $\frac{1}{4}$ inch	3.5 in.
2.....	Female.	12.25 "	6.8 "	behind fissure. $\frac{1}{8}$ inch	4 "
3.....	Male.	12.25 "	6.8 "	behind fissure.	4 "
4.....	Male.	14 "	7.7 "	Over fissure. $\frac{1}{8}$ inch	3.75 "
5.....	Male.	13 "	7.2 "	behind fissure.	3.75 "
6....	Male.	12.5 "	6.9 "	Over fissure.	3.75 "
7.....	Male.	12.5 "	6.9 "	Over fissure.	3.75 "
8.....	Female.	12.5 "	6.9 "	Over fissure.	3.5 "

In three (all male) the cyrtometer was absolutely correct.

In two (both female) correct in all but length.

In one (male) one-eighth inch behind fissure. Length correct.

In one (male) one-eighth inch behind fissure. One-fourth inch shorter than fissure.

In one (female) one-fourth inch behind fissure. One-fourth inch shorter than fissure.

In not one did the fissure of Rolando open into the fissure of Sylvius;³ neither was there found a convolution bridging the fissure of Rolando.⁴

Of the various methods proposed for locating the fissure of Rolando, I think Mr. Hare's the best, for these reasons:

First. The cyrtometer is quickly applied and easily retains its place. There are no planes to determine, no lines to draw, and but two landmarks are necessary.

Second. It is as correct as any method can be in the present fashion of heads. Its errors are small, and when they occur need not be feared. I would advise its use.

³ Turner. ⁴ Féré.

Books Received.

AMERICAN RESORTS, with Notes upon their Climates. By Bushrod W. James, A.M., M.D. Philadelphia and London, 1889. F. A. Davis.

EXTRA-UTERINE PREGNANCY—A Discussion. Reprinted from the Transactions of the American Association of Obstetricians and Gynæcologists: Philadelphia, Wm. J. Dornan, Printer, 1889.

PRACTICAL LESSONS IN NURSING DISEASES AND INJURIES OF THE EAR Their Prevention and Cure. By Chas. Henry Burnett, A.M., M.D. Philadelphia, J. B. Lippincott Co.

A TREATISE ON HEADACHE AND NEURALGIA. Including Spinal Irritation and a Disquisition on Normal and Morbid Sleep. By J. Leonard Corning, M.A., M.D. With an appendix: 'EYE STRAIN A CAUSE OF HEADACHE. By David Webster, M.D. Illustrated. Second edition. New York. E. B. Treat, 5 Cooper Union.

A TEXT-BOOK OF MENTAL DISEASE, with Special Reference to the Pathological Aspects of Insanity. By W. Beaver Lewis, L.R.C.P., M.R.C.S (London, Eng.: Philadelphia, P. Blakiston Son & Co., 1890.

SPINAL CONCUSSION—Surgically considered as a cause of spinal injury, and neurologically restricted to a certain symptom-group, for which is suggested the designation, Erichsen's Disease, as one form of the Traumatic Neuroses. By S. V. Clevenger, M.D. Philadelphia and London. F. A. Davis, publisher, 1889.

NOTICE.

THE AMERICAN NEUROLOGICAL ASSOCIATION.

The Council of the AMERICAN NEUROLOGICAL ASSOCIATION have decided that the Sixteenth Annual Meeting of the Association will be held at Philadelphia, Pa., on Wednesday, Thursday and Friday, June 4th, 5th and 6th, 1890.

There will be two daily sessions, one at 10.30 A. M., the other at 3.30 P. M.

DR. G. M. HAMMOND.

Secretary's Office,

58 WEST 45TH STREET.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

A CASE OF OPHTHALMIC MIGRAINE.¹

By J. CHALMERS DA COSTA, M.D.

THE association of diverse nervous phenomena with many cases of migraine has long been recognized by the profession.

Latham speaks of colored glimmerings occurring to the outer side of the visual field, with an inability to see some objects in the field.

Abercrombie compiled many observations of curious motor phenomena, spasmodic and paralytic.

Gowers speaks of aphasia being associated with migraine and numbness of the right side.

Da Costa speaks of its association with numbness and anæsthesia of an extremity.

Prony and Féré mention migraine linked with numbness of the hand and tongue, temporary aphasia, and epileptiform attacks.

Charcot points out hemianopsia and aphasia.

Weir Mitchell noted hallucinations.

Dr. Laundly, of London, has recorded a most remarkable case of recurrent migraine, in which during the attack there was paralysis of the third nerve of the left side, ptosis, absolute paralysis of the superior, inferior, and internal recti, pupillary dilatation, and paralysis of accommodation.

¹ Read before the Philadelphia Neurological Society, February 24, 1890.

Recovery after each attack was not absolute. The superior rectus became permanently paralyzed, and the other two recti weakened, and some degree of ptosis persisted.

Other observers have spoken of migraine being occasionally associated with perturbations or affections of taste, smell, hearing, loss of consciousness (partial or complete), vertigo, temporary hemiplegia, and transient blindness.

It is thus manifest that anomalous symptoms, if not frequent, are at least not unusual; but, in spite of this, I beg to present a case, probably not new to my auditors, but which to me was novel and peculiarly interesting.

A single woman, aged thirty-two, of a melancholy temperament and nervous diathesis; has passed the menopause. Family history is neurotic, the mother having had neuralgia and having been hypochondriacal. The father died of phthisis. Ever since her childhood she has had attacks of sick headache, coming on at irregular intervals, a month occasionally intervening. The attacks would terminate in vomiting, and were not connected with menstruation.

About two years ago the menses ceased, and the attacks of pain began to appear, with some degree of regularity, about every three weeks. The attacks are preceded for some hours by dull, unlocalized headache, anorexia, nausea and languor.

The eyes feel heavy and weak, and light is unpleasant. *Muscae volitantes* flit before the right eye, followed by colored spectra, flashes of light, and a scintillating scotoma. Intense paroxysms of lancinating pain occur in forehead, temple, and eye of the right side. There is marked photophobia, the eye is red, the pupil is dilated, and reacts slowly to light.

After some hours the paroxysms occur so frequently as to seem almost continuous, violent vomiting sets in, and the patient, apparently from exhaustion, falls into a profound sleep.

On several occasions, with the appearance of the scotoma, she has had numbness, pins and needles, and formation of right arm, lasting until termination of seizure, and accompanied by marked muscular weakness.

Again, anæsthesia of right arm has been observed, lasting for many hours.

Several times hemianopsia has replaced the scotoma, the right lateral half of each visual field being lost.

On one occasion the vomiting was not followed by sleep, and for many hours marked paraphasia existed.

To sum up :

Occasional transient aphasia ; transient hemianopsia ; numbness, anæsthesia, and muscular weakness in right arm, violent pain in ophthalmic division of the fifth nerve, scintillating scotoma and vomiting, occurring with more or less regularity, in a woman of middle age, of a *nervo-melancholy* temperament, and in poor general health.

The existence of such symptoms leads us to look for an explanation.

Dr. Living maintains emphatically that migraine and epilepsy are closely related.

An attack of migraine, he tells us, is due to an accumulation of nerve force and unstable nerve elements, the accumulated force reaching a high degree of tension, and exploding in a storm of pain.

According to this view, an explosion upon the motor sphere means epilepsy ; on the psychic sphere purely, epileptic mania ; and on the sensory sphere, neuralgia.

Living believes the fundamental cause of migraine to be "a primary and often hereditary vice of the nervous system," and the seat of this nerve instability to be in the optic thalami and parts between them and the roots of the vagi.

Dr. Stevens of New York, on the contrary, looks to reflex peripheral irritation as a cause of migraine, and insists that in most instances he finds this cause to be due to errors of refraction or accommodation.

Dr. Ringer forms a class of diseases, which he calls the explosive neuroses. This includes ordinary neuralgia, tetanus, asthma, epilepsy, migraine, epileptic mania, etc. These are due to a weakening of the nervous power of control, to a loss of resisting power, so that irritations which should cause impressions limited to small and definite areas, cause impressions which diffuse, spread out, flow over wide and often distant regions, producing symptoms according to the region attacked.

Take a case of ophthalmic migraine. The irritation causes primarily a flow of force from the nucleus of the fifth in connection with the ophthalmic division. As resistance

is weakened this force flows back to the nucleus of the pneumogastric, and vomiting occurs.

If the discharge is excessive in power, or if the resistance is much impaired it will flow to more distant points and produce symptoms of the most variable nature (sensory, motor, and psychic).

I would inquire of the Society as to the future of a patient with ophthalmic migraine, especially of this form. Is migraine ever a prodrome or early symptom of serious organic mischief.

Charcot says that the future of a subject of severe migraine is always uncertain. The attacks may recur for years and even pass away entirely, no other trouble being manifested, but in some cases it is a precursor of general paralysis of the insane.

Duchenne considers migraine a not unusual prodrome of tabes dorsalis, and Oppenheim of Berlin found it present in twelve tabetics out of eighty-five.

And finally Austie, noting that neuralgia might precede paralysis of a part, states his belief to be that neuralgia is the first expression of a condition which tends to become paralysis.

CLASSIFICATION OF MENTAL DISEASES.

The "Glasgow Medical Journal," February, 1890, quoting from the "Annales Médico-Psychologiques," September, 1889, states that the following classification was presented to the International Congress of Mental Medicine, held in Paris last August, and adopted as a basis for international statistics:

1. Mania, comprising acute delirium.
2. Melancholia.
3. Periodic insanity (double form, etc.).
4. Progressive systematized insanity.
5. Secondary dementia.
6. Organic and senile dementia.
7. General paralysis.
8. Neurotic insanities (hysteria, epilepsy, hypochondria, etc.).
9. Toxic insanities.
10. Moral and impulsive insanity.
11. Idiocy.

L. F. B.

REMARKS ON THE PATHOLOGY OF CHOREA.¹

By E. D. FISHER, M.D.

IN an article in the "Lancet," 1889, Dr. A. E. Yarrod describes in cases of acute chorea of rheumatic origin, an increase of connective tissue in the cerebral cortex. This pathological condition accords with the clinical aspect of the disease, acute or chronic, as one of the characteristic features of chorea is the increase of movement on any excitation of the will. In other words, call the cerebrum into action by concentration on any special motor act, and as a result motor disturbance ensues.

Another marked symptom is the constant motor disturbance, which ends only with sleep, a fact significantly pointing to the cortex cerebri as the primary seat of the lesion in chorea. We have here to do with a disease whose principal symptom is an affection of the motor apparatus. What is clearly seen is that we have a loss of the normal inhibitory action of the cerebrum.

The constant motor disturbance in some acute cases is due to the irritant effect of the connective tissue—that is, if we accept Yarrod's theory; and this is probably very often due to a rheumatic diathesis.

In chronic chorea we have a lesion resembling that of multiple sclerosis, although less coarse in character. Diller, in the "American Journal of the Medical Sciences," December, 1889, well defines it as a fine general sclerosis. We may have patches of degenerated nerve-tissue, the result of diseases of the vessels, causing anæmia and interference with the nutrition of nerve cells and fibres.

This leads to irregular stimulation of the motor tracts. We do not as a rule get actual paralysis nor indeed the increased reflexes following secondary degeneration in the

¹ Read before the New York Neurological Society, March 4, 1890.

cord. We almost always find, however, some paresis, and one of the symptoms present in multiple sclerosis, mental dullness. Arndt affirms that there is always some mental disturbance in chorea. In chronic cases it is especially marked, as also in hereditary chorea.

In Mercier's paper on "Inhibition," in "Brain," October, 1888, he compares the nervous system to an army under its different officers; the loss of any one of these heads leads to disorder in some of the various departments. This idea has always seemed to me too mechanical. In the nervous system we have to do with living organisms; their action or function is continuous, and dependent on their nutrition. The higher centres are subject to constant influence from without, through their lower centres and nerve-paths, and in this manner a constant relation or equilibrium of all the various parts of the nervous system is maintained. There is continuous action going on in the central ganglia, it is not only expended in the carrying out of some motor effect.

Let the peripheral stimuli become excessive, and these centres become exhausted, and as a result irregular efferent impulses are sent to the motor apparatus, as seen in the different occupation neuroses. I agree with him that no general or special inhibitory centre exists, but rather that the control or inhibition lies for each part in its corresponding centre.

An irritable condition of a centre, produced mechanically or through nutritional changes, may result in the so-called explosion of that centre, as in an epileptic seizure, or again, as in chorea, we may have continuous efferent impulses sent to the motor apparatus when no voluntary act is being carried out. When the lesion is more extreme, inhibition is still more affected, and purposive action only the more forcibly brings out the irregular motor effects.

It is possible, therefore, that lesions involving the nerve-tracts as well as the nerve-centres may be considered as causing chorea, thus allying it to multiple sclerosis; although when we remember that it is the muscles of specialized action—*i. e.*, of the hand—which are first and most

severely affected, it is probable that the primary lesion is in the cerebral centres controlling those parts.

In paralysis agitans we have a tremor which may be inhibited, and, if we accept Dr. Broadbent's theory of the seat of the lesion in this disease as lying in the muscular nerve-endings, we can readily understand the reason why this is so: the primary lesion points away from the cortex, whatever the secondary changes may be.

In acute chorea we find anæmia and vaso-motor changes more often present than any other condition.

Hanford, in "*Brain*," ———, 1889, quotes Dr. Dickinson as ascribing acute chorea to a widely spread hyperæmia of the nervous system, and in his own two cases he found, post-mortem, numerous hæmorrhages of the brain and cord with thrombosis and dilatation of the small vessels and capillaries. He considers the spinal cord, rather than the cerebral cortex, as the primary seat of the disease, as he believes the movements can be controlled in at least mild cases.

The connection of chorea and rheumatism is certainly more than accidental, but at the same time I do not believe it is as frequent as many writers would have us believe.

In chronic chorea we can look for atrophy of the cortex and degenerative changes in the nerve-tracts as a result of the hyperæmia and extravasations found in acute chorea. In support again of this theory I would refer to two cases reported by M. D. McLeod, in the "*Journal of Mental Sciences*," July, 1881, of two sisters, the chorea commencing, in the first, at the age of sixty; the autopsy revealed a cyst under the dura mater, over the left hemisphere, with flattening of the convolutions. The second sister was affected at the age of seventy, and the autopsy showed multiple tumors, with compression of the cortex. In these cases the compression and atrophy produced the chorea.

Dr. A. B. Ball, in an interesting paper on "*Thrombosis of the Cerebral Sinuses and Veins*," in the "*Transactions of the Association of American Physicians*," vol. iv., refers to this condition as occurring in chlorosis in young women, and reports several fatal cases with autopsies. The author

makes no reference to the occurrence of chorea in these cases, but that such a thrombosis may result in atrophy of the convolutions from permanent occlusion of the veins, entering the longitudinal sinus in the same manner as described by Gower, in cerebral spastic hemiplegia in children, seems to me very probable, although I cannot refer to any autopsies in proof of it.

Certain it is that in just such cases of chlorosis we find chorea unassociated with rheumatism or cardiac disease. The acute cases recover with the re-establishment of the circulation, while chronic chorea results from the atrophies referred to.

Acute chorea is as curable, therefore, as the conditions causing it, while in chronic chorea the prognosis depends on the character and extent of the lesion. The folly, therefore, of ascribing to the removal of a peripheral irritation, such as eye-strain, the possibility of effecting a cure in chronic chorea—that is, if we accept the pathology of it as just given—is apparent.

My paper has referred especially to the pathological changes in chronic chorea, and has emphasized the primary seat of the lesion as lying in the cortex cerebri.

UNILATERAL ABSENCE OF KNEE-JERK—PATHOLOGICAL FINDING IN A CASE.

Dr. A. Pick (*Arch. f. Psych.*, 1889, vol. xx) reports a case in which a diagnosis of tabes and dementia paralytica was made. The symptoms were: girdle sensation, retention of urine and fæces, slight paresis of the left facial nerve about the mouth, unsteady gait, tremor of the tongue, delusions of grandeur, etc. The knee-jerk was absent on the left side, and only to be elicited by reinforcement upon the right.

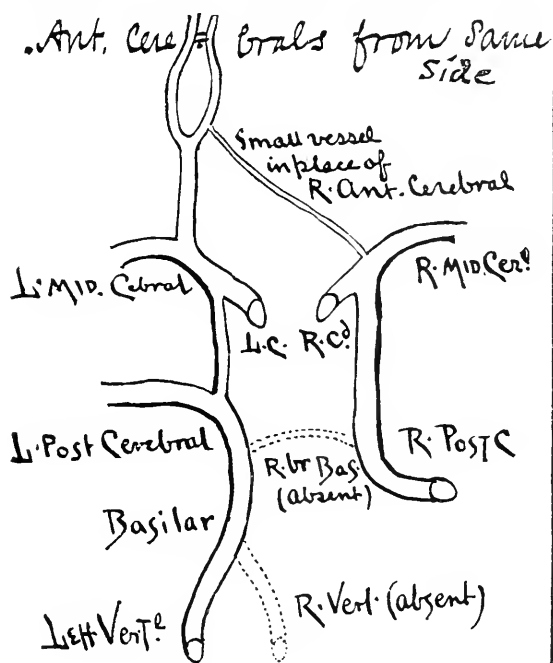
Autopsy: Chronic internal hæmorrhagic pachymeningitis, chronic inflammation with thickening of the other membranes, atrophy of the brain. Degeneration in the cord on the border of the columns of Goll and Burdach from the lower cervical region down. In the upper dorsal region there was on the left also a narrower stripe next to the posterior horn, and on the right a similar one in the middle of the column of Burdach. In the lower dorsal and in the lumbar portions of the cord, on the left side, the posterior root zone was affected.

F. P.

SPECIMEN SHOWING ANOMALY OF THE CIRCLE OF WILLIS.¹

By JAMES HENDRIE LLOYD, M.D.,

THE specimen was taken from an insane man, who died in the Philadelphia Hospital—a case of chronic mania. In the large number of brain-dissections which I have seen or conducted, this is the first time I have met with an anomaly of the circle of Willis, although such anomalies



are not uncommon. This anomaly under some circumstances might be of clinical or pathological interest, and will be best illustrated by a diagram. Ordinarily the two

¹ Presented to the Philadelphia Neurological Society, January 27, 1890.

vertebral arteries unite to form the basilar ; in this case the right vertebral artery did not exist. The basilar artery was a continuation of the vertebral artery of the left side. It sent off as usual the posterior cerebral artery. The posterior communicating artery on the left side was a very large trunk ; in fact, this was one large continuous vessel from the foramen magnum. The left internal carotid then divided into the middle cerebral and the anterior cerebral. The anterior cerebral arteries of both sides sprang from this one trunk. Practically more than one-half of the brain was supplied from this one side. On the other side the carotid gave off as usual the posterior communicating artery, which is a large trunk, in this case practically continuous with the posterior cerebral artery. The branch of the basilar, which as a rule forms the posterior cerebral artery, is here small, rudimentary, and probably non-pervious, as is also the branch of the carotid, which ordinarily forms the anterior cerebral.

This anomaly is described by Duret. One singular thing is that the branching of the posterior cerebral from the carotid generally takes place on the right side. The vertebral artery of the left side is usually the largest, and when only one is present it is apt to be the artery of the left side. The only apparent reason for this is that the left vertebral artery derives its supply more directly from the subclavian than does the right vertebral. What pathological interest this anomaly has I cannot say. Embolism in such a case might give rise to some peculiar symptoms ; but I doubt if the condition could be recognized in life.

THE FUNCTIONS OF THE BRAIN.

The doctrines of the Italian school are embodied in the first part of a paper (fifty pages) by Jules Soury in the November number of the "*Archives de Neurologie*." The cortical centres for cutaneous and muscular sensibility and for voluntary movements are considered.

HYSTERO-EPILEPSY IN BOYS. (*Ibid.*)

The history of three cases forms a contribution to the study of this subject, by Bourneville and P. Sollier. L.F.B.

FUNCTIONAL NERVOUS DISEASES AND THEIR RELATIONS TO GASTRO-INTESTINAL DERANGEMENTS.¹

BY W. H. THOMSON, M.D.,

Prof. Materia Medica, University Med. College, N. Y.; Physician to Bellevue and Roosevelt
Hospitals.

AS a teacher of the materia medica it has long been my practice to divide all medicines proper into two great classes, namely, the organic and the functional. The organic medicines are those whose special remedial action can never be secured by one dose, but only by the slowly cumulative effects of many repeated doses. The functional medicines, on the other hand, are those whose whole specific action is obtained by one dose, and no repeated doses of them ever effect more than did the first dose. Iron given for anæmia or mercury for syphilis are examples of the organic medicines: opium and belladonna are examples of the functional medicines. The organic medicines are given for the diseases or morbid conditions themselves and not for their symptoms. The functional medicines are given for the symptoms and for nothing else. If, therefore, potassium iodide relieves the pain of a syphilitic node, it is because it removes the node itself. If opium, on the other hand, relieves the same pain, the node still remains to renew the pain so soon as the effect of the dose has worn off. The organic medicines should not produce any symptoms of their own. If they do, they are not producing their remedial effects. The patient should not know that he is taking anything, except by signs of returning health. The functional medicines, on the contrary, do nothing but produce symptoms. Organic medicines show no effects, that is, not the effects for which

¹ Read before the County Medical Society of New York, Jan. 27, 1890.

they are given as remedies, except in states of disease. Iron does not increase the strength of a healthy man, and no healthy man will show any beneficial effects from mercury such as a syphilitic person does. But functional medicines show the same symptoms in health as in disease. Strychnia does not need an invalid to illustrate its properties, and an emetic will act in its proper dose on the most normal stomach.

Time will not allow still other contrast, to be presented between these two classes of medicines, but what has been adduced suffices to show that there is this fundamental difference between them, namely, that by the slow cumulative action of the organic medicines we produce organic changes in the nutrition of the body, either in its fluids or in its solid tissues. By the functional medicines we produce no organic change whatever. By repeated dosing with them we only do over and over again what we did with the first dose. Functional medicines, therefore, never cure any disease. The most they can ever do is occasionally to break a nervous habit. Hence, as the neurotics belong to the class of functional medicines, no true nervous disease can be cured by a neurotic. All a neurotic does in any nervous disease is to relieve some symptom of that disease, leaving the disease itself the same as before. A man may have taken a thousand doses of stramonium for his asthma, but the thousandth dose can do no more than the first dose did, namely, relieve the symptom spasm, but in no way modify the disease (asthma) itself.

The only difficulty which I have experienced in demonstrating these principles has arisen from a slowness on the part of some minds to recognize what the difference between the function of an organ and the organ itself is. How an agent, for example, like *veratrum viride*, or like *digitalis*, can act on the function of the heart and not on the heart itself, is to them puzzling. But this should be no more puzzling than the common fact that a man may act on his brain for years with powerful doses of opium or of tobacco and yet not affect that organ itself enough to furnish the least clue for the most skilled microscopist to distinguish

the brain of an opium or tobacco smoker from that of one who never took either of these poisons. For this difficulty the most commonly advanced hypothesis is that specific organic changes doubtless are caused in nerve tissue by these strong poisons, but that these are of such a molecular character that we have not yet attained to the requisite methods for their detection. That there must be *some* change induced is taken for granted as almost a self-evident proposition resulting from the supposed necessity that for every manifestation of nerve function there must be a corresponding physical basis in nerve matter. Moreover, this conception seems to be further supported by the undoubted fact that structural changes in any organ, including nervous organs, always manifest corresponding functional derangements, and therefore we may suppose the converse to be also true, namely, that functional derangements always have their corresponding structural changes.

But a little reflection on what function in distinction from structure really is may suffice to show that function may be either deranged or wholly arrested without structure being involved at all. For the function of an organ consists in the work that it does and in that alone. But for the working of any organ, or for that matter for the working of any mechanism, not only is structure needed, but something *plus* structure. Respiration, for example, is the function of the lungs, secretion that of a gland. Now we can either stimulate or decrease that working without affecting even molecularly the texture of the lung itself or of the gland, just as we can increase the working of a steam-engine by adding more coal, or stop it by putting the fire out, without in either case affecting the structure of the iron mechanism itself. In an oil-lamp also we may have its function of giving light much disturbed by adulteration of its oil, from diminution of its light to veritable explosion, without its own mechanism, either in its wick or in any other part of its apparatus, having anything wrong about it. It is just as true in this case that any damage to the structure of the lamp may correspondingly affect its light-giving function, but that does not prove that whenever it is disturbed in its

lighting power some alteration must have happened in its structural parts.

The lamp needs for its function good structure first, but also something else. The nerve-cell likewise needs normal specially differentiated protoplasm first, but also something else, and which something else may be so deficient or deranged that the cell function will be correspondingly abnormal, no matter how normal the protoplasm itself be.

I do not see why, therefore, these illustrations do not hold good in the case of functional derangements in the *living* mechanism. We have a number of affections which in the last analysis seem to be wholly disorders of function. We say last analysis, because no methods yet discovered of investigation carry us farther than the determination of a functional derangement. Every known mode of examining structure fails of demonstrating in them the least characteristic departure from structural integrity; and yet to many minds it seems unphilosophical to rest content with the term "functional." They insist that every functional derangement must have a corresponding anatomical change to which it is due and which will account for it, and they look forward to the time when the progress of our knowledge will banish the term functional from medical terminology altogether by giving us in the case of epilepsy, hysteria, migraine and other now-named functional affections the proper anatomical designation for them. But this view arises wholly from the conception that nerve-cells work spontaneously by virtue of their organization, and therefore that when they seem to work abnormally something must have gone wrong with the arrangement of their physical molecules. It seems to be overlooked that without a constant supply of a material or materials for their working, nerve-cells must remain as inactive as a gun without its powder. If the powder be too small in quantity, or defective in making, or has been wet, all the unsatisfactory working of the gun therefore was from no fault in *it*, and why may not a nerve-cell work badly from no fault in it, but solely from deficient or poisoned function pabulum?

This question is not one of theoretical, but of practical

importance. If every functional nervous disease is really due to a definite morbid change in either nerve-cell or nerve-fibre, then we must continue to search in nerve-cell or in nerve-fibre for that change and for its causation. If, however, such nervous affections are not at all to be traced to faults of structure, but rather to disordered sources of nerve energy, then we must look *elsewhere* than to the nervous system for both the causation and for the remedying of these disorders. But in examining the conditions under which manifestations of energy take place, we are met at the outset by the fact that the relations of energy to structure are in no instance yet known to science, not even in the so-called mechanical forces. Our knowledge of such relations is purely empirical. Why electricity, for example, will traverse an iron rod but not a glass rod, we neither know nor can explain. It does not seem philosophical, therefore, to expect that in the case of nerve-function we are going to discover its structural relations any easier, and certainly not in disordered function, when we have not the slightest clue to the physical basis of a normal sensation, or of a motor impulse or of a thought. The structure of no mechanism for the utilization of energy ever gives the least indication, to one who inspects it, of what the thing is which energizes it. All he can do is to note its mode of working. And this is also all that one can do in those disorders of nerve-function which we can induce artificially by administering functional medicines. What it is that hydrocyanic acid does to a group of cells in the medulla we can only tell by describing the resultant disturbances of nerve-working, and there we must stop. But equally in those functional disturbances which cause a convulsion or a neuralgia, all we can do is to observe rather than to explain. We are not even assisted by noting how much the structure is worn by its working. Liebig erroneously thought that the energy of the muscle-cell was generated by its own self-expenditure, and hence that muscular exertion might be measured by the amount of urea formed, and there are some who still speak of the nerve-cell generating its force by using itself up in the process. But we now

know that we might as well explain the flight of a bullet by noting how much the gun was worn by its discharge.

Regarding our subject, therefore, from the empirical standpoint of clinical observation, which is the only practical one, the question then arises, are there any features which distinguish supposed functional nervous diseases as a class so plainly that we can conclude from these features that they are distinct in kind from organic nervous diseases? If so, what is the significance of those distinctions, and to what conclusions do they point as to the essential characters of these disorders?

There is one distinction which has always seemed to me of fundamental import, and it is this: that no structural or organic nervous disease is ever truly *intermittent*. A hemiplegic may show a greater degree of weakness one day than another, but he is always hemiplegic nevertheless. A patient with fully developed locomotor ataxia may vary considerably in his symptoms from day to day, but never is he so much better that no one would be able to detect a sign of disease in him. And so with any of the other nervous diseases characterized by structural changes in nerve-tissue. Indeed it is inconceivable how it can be otherwise. A positive loss or degeneration in nerve-cells or nerve-fibres must show as closely corresponding defects in the use of those textures as in the case of loss or degeneration in muscle or bone. But quite otherwise is it with true functional nervous diseases. The most skilled nervous specialist may be wholly unable to suspect that a man whom he meets in company will within an hour terrify the room full of people by falling in a violent epileptic fit; or that the vivacious lady near him will be prostrated the next day with a severe sick-headache. Nothing, in fact, seems more complete than the disappearance of all signs of any disease, nervous or otherwise, in many patients with pronounced and definite functional nervous affections during the intervals between their attacks. We might as well expect to detect by our means of exploration whether a given person be avaricious or generous as whether, in many typical cases of functional disorders, they are subject to

convulsive or to neuralgic paroxysms, and it is probable that anatomy will never be able to tell us how these fits happen according to structural nervous changes, until it is also able to give us the structural anatomy of cupidity or of benevolence.

But as it is difficult to imagine how an organic nervous disease can be ever truly intermittent, so it follows that a truly intermittent disease cannot have an organic basis in the nervous system. This proposition is in no way invalidated by citing those cases of epilepsy which accompany an organic lesion within the cranium, such as a pachymeningitis, syphilitic or traumatic, and which in some instances have been relieved by a surgical operation. All that can be said for the causation of the epilepsy in such instances is that any abnormal or simply unusual afferent impression, whether proceeding from an intercranial or from an extracranial focus, may cause epilepsy; as, for example, a case of severe and protracted epilepsy which supervened upon a wound of a testicle and which was cured by the removal of that testicle. Every afferent impression to which the affected nerve-centres are wholly unaccustomed may be dangerous in this way, as witness the numerous published cases of fatal status epilepticus caused by injections for washing out the pleura and also after washing out the stomach. In fact organic changes within the skull, if they occasion epilepsy, do so on just the same principle that a tapeworm in the intestine may do likewise, and they do not, therefore, afford any indication that this functional disease has at any time a true specific organic basis.

If, therefore, we have any supposed functional nervous disease, whose manifestations are continuous, and not intermittent, the presumption becomes strong that it has an organic basis, even though such has not been yet demonstrated. Paralysis agitans, for example, comes within this category, and recent investigations seem to render this surmise correct. Chorea, on the other hand, will, we think, turn out to be an exception which proves the rule. The presence of a toxic element in the blood, rather than of an organic change in the nervous system, is becoming more

and more probable as the commonest cause of this affection. The great frequency of carditis in choreics, as shown by Osler, even though they have never given a history of rheumatic seizures, strongly supports the view, which I have long held, that the rheumatic poison may, in early life, manifest itself in no other way than by nervous symptoms. In the case of gout, we know that its poison often ceases in the adult to develop articular symptoms and to attack nerve-centres instead. I have a patient who can date the particular day, more than ten years ago, on which that happened after a profound nervous impression, and he has been troubled ever since with the most intractable and varied functional nervous disturbances. That the nervous system of children may therefore be specially susceptible to the rheumatic poison, more so than their articular textures, has nothing inherently improbable in it, especially as with numbers of them we find the heart also more readily affected than the joints. In common with many other observers have I been repeatedly struck with the proneness of some children to develop carditis when other signs of the presence of rheumatism, including arthritis, were but little pronounced, and this fact alone takes away much of the value of some statistics which have been published of the occurrence of chorea without rheumatic antecedents. The case indeed just cited, of the transference of gout from the joints to the nervous system from nervous shock, suggests an analogy with the often alleged origin of chorea from fright.

In severe acute functional nervous diseases, the profession, following the clue afforded by hydrophobia, has been led to look elsewhere than to the nervous system for the specific cause, and in the case of tetanus has found it. But on the same principle we ought to search likewise for the cause of Landry's paralysis. Anyone under the domination of the anatomical theory, who may see a case of this formidable disease and then watch the steady but speedy ascent of the palsy until bulbar symptoms successively develop, will feel sure that a fearfully rapid disorganization of nerve-centres is progressing under his observation. But

after death nothing is found in either the peripheric or in the centric divisions of the nervous system that tells any consistent story. One or two isolated observations of limited peripheric changes comprise all the trustworthy nervous pathological anatomy of acute ascending paralysis. Other examinations by most competent investigators have shown no more certain indications how death was caused than examination of the medulla shows how hydrocyanic acid kills. On the other hand, I once had a patient with that toxic paralysis which follows diphtheria, die with many of the typical symptoms of Landry's paralysis. And it is not without significance that in nearly every autopsy of this affection, enlargement of the spleen, and often of the mesenteric glands, has been found, suggestive of a poison in the blood, rather than of an organic nervous mischief as an explanation of the mortal issue.

If, therefore, such acute functional affections, severe enough to be terribly fatal, run their course without a single characteristic change in nerve tissue, we can scarcely expect this change in the chronic varieties of functional diseases which have their long intervals of complete cessation. During these intervals, the nervous system goes on with as little sign of disturbance or of interference as it does in a man who gets intoxicated with alcohol only once every few weeks or so. But while this great feature of intermittency is utterly at variance with any theory of structural alteration that can be framed, it is not incompatible with certain facts which the study of functional medicine affords. By neurotics we can artificially induce some close imitations of functional nervous diseases, and cause delirium, convulsions, comas, neuralgias, paralyses and all intermediate symptoms of the kind, and note these symptoms disappear in about the time and order that a functional nervous attack develops and declines. The feature of intermittency would then correspond to the intervals of intoxication in this respect, namely: that with analogous periodic doses of opium, for example, we would have recurrent attacks of functional derangements, giving just the same symptoms each time, with no new element developing,

just as functional nervous diseases recur for years without indicating that ominous feature of progressive extension which goes with most organic neuroses.

So far, the analogy is good enough, but it may be well urged that the toxic theory of functional neuroses can hardly hold good for those cases which are associated with a palpable organic focus of irritation, as epilepsy from malformation of the skull, or from a pachymeningitis, or in a reflex case from a renal calculus, etc. Is it to be supposed that such permanent exciting causes of the convulsions act by generating poisons? To this it may be replied that the problem of intermittent derangements with permanent exciting causes is no better explained by any other theory than that of a concurrent toxic influence, for the permanent irritant cannot of itself be enough to cause the convulsion, else the convulsion would neither intermit nor cease, but be as continuous as the irritant is. There is in every such case, the irritant and something else, and not till both act simultaneously does a convulsion occur. It is, therefore, just as easy to explain the case by saying that the permanent irritation acts by developing an idiosyncrasy to the operation of certain recurrent poisons in the blood which did not exist before the irritation. This is by no means a fanciful hypothesis, for the relation of the subject of idiosyncrasies to neurotics can be shown to have a much closer relation to the phenomena of functional neuroses than any anatomical facts can. Without the slightest indication to forewarn us, we may kill a patient with an insignificant dose of chloral or of antipyrine, and in a less degree we are constantly annoyed with the unexpected and undesired response of the nerve-centres of individuals to some of the most commonly prescribed drugs. This extraordinary susceptibility to certain drug poisons is almost always a limited affair, that is, limited to certain nerve-centres in that particular individual, so that one person's idiosyncrasy is against chloral but not against morphia, or vice versa. What it is that weakens the resisting power of certain centres to certain poisons only we cannot tell, but it is quite conceivable that such a weakness may be set up by a permanent sensory

irritation which may cause a toxic influence to be as operative in causing a convulsion as a single indigestible article in the stomach has been known to develop the first attack of uræmic asthma.

If insight into the origin of functional nervous diseases, therefore, is not to be expected from an investigation of their structural relations, in what direction are we to look for more satisfactory indications of their true nature, and thereby of their treatment? In reply, we would begin by a single clinical illustration:

A lady consulted me for very pronounced and severe symptoms of Graves' Disease, except that she had only moderate enlargement of the thyroid and but slight exophthalmos. She had, however, an exceedingly rapid pulse, tumultuous heart action, great loss of flesh, and *persistent diarrhœa*. She did not improve under a varied medication, but at once began, literally, to get well when she was put on an exclusive diet of fermented milk. After some months, she tired of the milk and resumed ordinary diet. Her old symptoms progressively returned. She took up the milk diet again, and at once greatly improved again. She then left off the milk, and though the symptoms once more recurred with great severity, yet she could not be persuaded to resume the milk until a short time before her death. Now, it is difficult to resist the inference that both the development of Graves' disease in her case was, for a time at least, a matter of diet—but what does that fact imply?

There may be (I only say there may be) an explanation in the view that a poison was generated in her intestines during her maldigestion which acted as a vaso-motor paralyzer when absorbed into the blood. This poison may have been exceptionally active in her case either from its specific nature, or because her liver, owing to some derangement, did not destroy the absorbed poison as it should, or lastly, because her kidneys did not eliminate it as they should. Room for speculation there certainly is, but the fact still remains—diet had more control over her disease than anything else had. Here a recent observation from a series of experiments by Charrin and Roger may have some bearing.

They found that the urine of a man fed upon an exclusively milk diet lost the toxic properties of normal urine when injected into rabbits, guinea pigs, and dogs indifferently in great the proportion of one to ten. (La Semaine Médicale, 1888.)

That decompositions are constantly occurring in our digestive laboratory in the healthiest state of the body, which yet are accompanied by the formation of definite and virulent poisons, is one of the most important discoveries of modern science. As Dr. Lannder Brunton remarks,¹ "We may now indeed regard alkaloids as products of albuminous decomposition, whether their albuminous precursor be contained in the cells of plants and altered during the process of growth, or whether the albuminous substances undergo decomposition outside or inside the animal body, or by processes of digestion. Thus the poisonous alkaloid muscarine, which had only been known as obtainable from a plant, the *Agaricus muscarius*, has been discovered by Brieger to be a product of the decomposition of fish. A considerable production of alkaloids takes place in the intestines, both when the digestive processes are normal and more especially when they are disordered; at the same time, alkaloids are being formed in the muscles and possibly in other tissues. Were all the alkaloids to be retained in the body, poisoning would undoubtedly ensue, and Bouchard considers that the alkaloid formed in the intestine of a healthy man in twenty-four hours would be sufficient to kill him if they were all absorbed and excretion stopped." That these formations of alkaloidal poisons are in many instances due to the activity of specific bacteria is also demonstrated. It is thus that indol is produced out of the products of pancreatic digestion, and then sent to the kidneys to excrete as indican, as shown by Brieger. The same has demonstrated by Thudichum to be the source of the coloring principles of the urine, first formed in the intestine and then sent to the kidneys for excretion. (Lancet, Dec., 1889.) It has even been proposed to use these facts to determine

¹ Pharm and Tox., p. 100, 89.

the degree in which the kidneys have been damaged in chronic Bright's disease, by noting how much less toxic Bright's disease urine is than normal urine, as an indication of the failing power of the kidneys to eliminate the poisons that come to it from the great digestive laboratory. Thus, according to Bouchard's modification of the original ratio of Feltz and Ritter, the urine of a healthy person injected into a rabbit's vein kills in the proportion of 50 grms. per kilogram of weight of the rabbit's body. But that of Bright's disease subjects is tolerated in much larger doses. Dieulafoy mentions a rabbit of 2 kilograms weight for which the toxic dose of healthy urine would be 100 grms. but which showed no discomfort until 260 grms. of the urine of a patient with Bright's disease had been injected, and even then recovered ("Lancet," June 4, 1887.)

The constant production during healthy life of these animal poisons as they can be gauged by their elimination in the urine, is shown even by their periodic variability. Thus Bouchard, ("sur la variations de la toxicité urinaire pendant la veille et pendant la sommeil." *Gaz. Hebdomadaire*, April, 1886.) shows that if the day be divided into three periods of eight hours each, the proportional quantities of poison excreted are: asleep, 3; early waking period, 7; late waking period, 5. The urine after sleeping and waking hours also differs qualitatively as well as quantitatively. The alkaloid of the urine of sleep is *convulsive*, that of the waking urine *narcotic*. Fasting increases the toxicity of the urine. Labor and increased respiration of fresh air greatly diminishes the toxicity. We would by such facts seem to have some clue to the nocturnal character of some cases of epilepsy, also of attacks of asthma, gout, etc., during the later hours of the night. But the chief interest of these discoveries lies in the illustration which they afford of the continued liability throughout life, of the nervous system to causes of the most serious functional derangements from the uninterrupted formation in all the processes of life of active functional poisons which would operate immediately were the means provided for their elimination or destruction to become disordered.

This subject, however is so extensive that we are precluded by want of time to allude to more than a few illustrative examples. Thus one of the most suggestive formations of a poison in the body consists of the allied poisons choline, neurine and muscarine, the latter two being extremely virulent. Now choline, which has been found by Brieger to be such a common product of putrefaction, has been found by numerous investigators (Boehm, Schoeff, H. Griers, Jahns, *et al.*) very extensively in leguminous plants, as beans and lentils, in peanuts, cottonseed, hops, and therefore in beer, besides in various edible fungi. By a single substitution of a molecule of water choline is turned into neurine, a substance which has been stated by Liebrich ("Untersuch. uber Ptomaine," I., p. 32) to exist only in brain matter, but Prof. Schmidt ("Pharm. Rundschau," 1887, p. 266) has shown that when hydrochloride of choline is allowed to stay in contact with blood for some time at a temperature 30-35 C. it is converted into neurine, while Wurz ("Bielstein's Org. Chem." p. 402) has shown how choline by treatment with an acid is converted into muscarine, the alkaloid found, as stated above, by Brieger in fish. In this connection the valuable researches of Knorr ("Pharm. Zeitung," p. 366) on the molecular constitution of morphine, are interesting, as he shows the very close relationship of the latter to choline.²

These facts at least demonstrate that we do not have to go far to find the explanation of every symptom of functional neuroses when the system is constantly producing the agents which would inevitably occasion them all without exception, according to the varying conditions of its functional activity. Headache, muscular languor, diarrhœa, depression of the heart or palpitation, depression of the spirits, and convulsions are among the toxic symptoms, and moreover, like our familiar drug poisons, these body-generated alkaloids have their relative affinities for different

² For an exhaustive account of this subject consult the able and scientific paper of Prof. F. B. Power and Jacob Cambier, of the University of Wisconsin, on their "Isolation of Choline from the Bark of the Common Locust or False Acacia," Supplement "Scientific American," Mar. 15-22, 189c.

nerve-functions as special and as characteristic as any of the agents of our *materia medica*. In fact, so close is the resemblance that they have often given rise to disputes in medico-legal cases. Thus, in the celebrated Brandes-Krebs trial, two chemists obtained from the cadaver in addition to arsenic, an alkaloid which they pronounced to be coniine, but which Otto proved only to be closely analogous to coniine and also to nicotine. Brouardel and Bautney found in the body of a woman who died from eating a stuffed goose a body which gave the odor of coniine and the same reactions of that poison with gold chloride and potassium iodide. In a criminal prosecution at Verona, Ciotta obtained from the exhumed but only slightly decomposed body, an alkaloid which he pronounced as identical with strychnine, until this identity was disproved by Selmi. So also, have morphine-like substances, others closely resembling atropine, others like digitalis, been found in similar toxic cases. ("Vaughan on Ptomaines and Leucomaines," p. 110, sg.)

Against this auto-infection or self-poisoning we seem to be protected in great part by the liver. It is fully demonstrated now that the liver possesses a two-fold power, to prevent poisons entering by the portal vein from passing into the general circulation, for it turns back some and destroys others. Some poisons, such as curare, are sent back in large part by the bile, but with others it either decomposes them altogether or renders them in large part innocuous, for double the quantity of strychnia, veratria or morphia is required to kill an animal if injected into the portal vein as would be sufficient if injected into the jugular vein, while no less than three times the quantity of curare is requisite (Brunton op. lit.) The remainder seems to be sent to the kidneys for elimination.

But a greater preservative still remains in the natural digestive secretions of the alimentary canal itself. In the case of the gastric juice this was shown long ago by De Haen, but the same fact holds true of the intestinal digestive juices that no better antiseptics can be found. This double function of the digestive secretions is too often over-

looked, namely: that they not only digest but preserve. Any suspension or perversion of normal digestive secretion therefore, **at once** raises the danger of poisoning from the ready perversion of the contents of the canal to undergo fermentation when the digestive juices are deficient. Often the resulting catarrh of the intestines from the local irritation of such fermentation leads to the flux of summer diarrhœa, which to a certain extent is therefore beneficial rather than harmful. But on the other hand, this danger of auto-infection is one of the commonest attendants of fever from the rapidity with which the fever process arrests all gland secretion, including the alimentary secretions, and no one who watches the symptoms of typhoid fever can fail to note how many of them resemble those of toxic infection. As we might expect, therefore, the urine has been found in fever, especially in typhoid, (Bouchard, Lepine, Guerin, Brunton) to be much more toxic than normal urine. Mr. A. P. Luff, Lecturer on Toxicology, St. Mary's Hospital, London, ("Brit. Med. Jour.," July 27, 1889) claims to have found a distinct alkaloid in typhoid fever and another in scarlet fever.

Closely allied to the genesis of functional diseases are the problems connected with those varied morbid states of the blood to which the general term anæmia is given, and in this connection, we can only refer to the admirable lectures on the "Pathology of Pernicious Anæmia," by Dr. W. Hunter, which form the most important recent contribution to medicine by British investigators. ("Lancet," Sept. 22, 29; Oct. 6, 1888.) After demonstrating that this is a specific disease on account of its showing a characteristic structural lesion, namely: the deposit of iron in the liver cells, he says: "In pernicious anæmia the seat of disintegration is chiefly the portal circulation, more especially that portion of it contained within the spleen and liver, and the destruction is effected by the action of certain poisonous agents, probably of a cadaveric nature, absorbed from the intestinal tract." This hypothesis is rendered all the more probable by the febrile character of this affection which has always served with me to distinguish this from other forms of seri-

ous anæmia with which it is often confounded. In later communications published in "The Practitioner," (see also "Lancet," Jan. 11, 1890) Dr. Hunter adduces still further confirmations of the view that pernicious anæmia is due to a hæmolytic process induced by some ferment in the gastrointestinal tract by observations on the excretion of pathological urobilin, of blood pigment, and of urine in this disease. In a typical case which he examined, he draws attention to the exceedingly high color of the urine although the specific gravity remained low. Bile pigments were not the cause of this coloration, for spectroscopic examination showed it due to that form of pathological urobilin which MacMunn proved to be distinct from the urobilin of normal urine and to be formed by excessive elimination of bile into the intestine. Dr. Hunter then instituted an inquiry into the excretion of iron in the urine in health and in disease, and he estimates the daily health average as from 3 to 5 millegrammes. He found that the administration of iron by the mouth hardly, if at all, increased the amount of iron excreted. In one observation in health the iron excreted amounted to 5.65 mm., in one case of chlorosis to 1.71 mm., in another 1.96, and in a third 1.61, considerably lower than the average but consonant with the diminished richness of the blood in hæmoglobin characteristic of this affection. But in the case of pernicious anæmia, the daily average of iron excreted for about three weeks in the urine amounted to the great figure 32.26 millegrammes.

In the afebrile disease, chlorosis, on the other hand, many modern investigators ascribe the blood-change to absorption of poisons from retained fecal accumulations. Sir Andrew Clark is such an advocate of this theory that he proposes to term chlorosis, fecal anæmia. There are some facts, however, in the genesis of this disease which seem to me to lie back of the constipation of chlorotics, and which have an important bearing on functional nervous diseases as well. In not a few conditions we seem to have an illustration of a vicious circle of interacting causes of disorders of digestion, beginning with a reflex nervous irritation suspending or perverting the digestive secretions and also

paralyzing the intestinal movements, while these effects in turn become causes of the formation and the retention of excrementitious matters, and thus of auto-infection. When I first entered on the practice of medicine I met a remarkable instance of the kind in consultation with two medical friends, who also called in the late Dr. White, of Buffalo, professor of obstetrics in the Bellevue Hospital Medical College, New York. The particulars of the case I published in the "Transactions of the New York State Medical Society," for the year 1867, p. 148. The patient, being a girl, aged seventeen, who, after a suppression of the menses, induced by a wetting in a cold shower, had first all the symptoms of intestinal obstruction with repeated fecal vomiting, but with no pain. By the administration of active purgatives some scanty movements would be obtained; but though calomel (with jalap) was freely given, yet the dejecta were uniformly so white as to resemble lime plaster. (We now know that this absence of color in the fæces denoted suppression of the pancreatic as well as of the biliary reactions.) If purgatives were not given, the constipation would continue, soon to be followed by sterco-raceous vomiting. Ere long, however, a new train of symptoms set in. First, the urine was suddenly suppressed, and the death of the patient was daily anticipated; but, instead of that, a profuse salivation occurred, amounting to about three pints daily, accompanied by a copious flow of tears, not caused by any emotion whatever. In fact, the mind of the patient was remarkably clear and composed from first to last, nor did she ever present a single symptom which could be properly termed hysterical. This combined salivation continued for about three days, and then ceased, whereupon the kidneys resumed their proper action for a few days more, and then stopped again, when immediately the saliva and the tears began to flow as freely as before. This alternation between these widely separated glands finally became a regular feature of the case, the change from one to the other sometimes occurring as often as on alternate days until death took place, in a little over two months from its first occurrence. Meanwhile the sterco-

raceous vomiting similarly alternated with small white alvine discharges, and the stomach refused to retain anything, so that life was maintained by nutritive enemata.

Now, in this case, and in others similar to be found in medical literature, we have but an extreme illustration of the power of reflected irritation from pelvic nerves to derange both the whole series of the alimentary secretions and of the gastric intestinal movements, yet not differing except in degree from the common derangements of the early months of pregnancy. But just such influences may precede, and as I believe actually do precede, the development of the hysterical status. Why is it not a probable deduction that in the usually perverted and disordered intestinal digestion of hysterical patients we may have all the conditions needed for a veritable intoxication of the blood with functional poisons to which, rather than to an occult fault in nerve-centres, the symptoms of the disease are due? Certainly, since I have treated hysteria with that conception in mind, by repeated purgation and the administration of intestinal antiseptics, I have had, as I think, much better results than by dosing them with many of the old-fashioned antispasmodics.

The same I can say of my treatment, in the main, of migraine. The frequent sallow complexion and the high-colored urine of these patients, on the subsidence of an attack of sick-headache, not to mention the early supervention of fermentative eructations in this affection, long ago led me to look upon the majority of these cases, not as instances of "nervous storms," but of cases of imperfect digestion; and it is a question whether most of our reputed remedies for this form of headache do not act chiefly by virtue of their antiseptic properties. The fact that many of these cases present a history of marked hereditary predisposition rather favors the toxic theory, for both rheumatism and gout are equally characterized by heredity. The phenomenon of periodic explosion of the attacks, however, which this affection shows in common with some cases of epilepsy, is closely analogous with the occasional action of

some functional poisons. For a long time I was inclined to be incredulous about the cumulative property of digitalis, simply because, like all incredulity, mine was based on nothing but the failure of my own experience to furnish a case, though I had prescribed digitalis largely. But one patient with phthisis, to whom I had given this drug in divided doses for about a week, banished my doubts ever after by a veritable explosion of the symptoms of that poison such as I will not soon forget. The same development is said to occur occasionally with strychnia. But the most marked illustration of this property of neurotics I had from a prescription, which I used, some ten years ago, quite extensively, in both hospital and private practice, for the reduction of temperature in phthisis, and which consisted of two drops of the tincture of aconite-root, with one drop each of the tincture of veratrum viride and of the fluid ext. gelsemin., given three times a day. It seemed to answer the indication very well during some two years' trial, but I was obliged to abandon it, owing to sudden and alarming symptoms of collapse occurring in five cases, after some two weeks' or so administration, three of them in the Charity Hospital and two in the Roosevelt Hospital. After recovery from these prostrations, however, the temperature remained down for prolonged periods in each of the patients, in no case less than two weeks and in one for three months.

I had intended to speak fully on the bearing of these considerations on the treatment of epilepsy, but time fails me now, and I will have to defer that to another opportunity. Of course, with such a great variety of afferent exciting causes to induce the attacks, one cannot claim that epilepsy can be ascribed chiefly to infection by functional poisons in more than in a certain proportion of cases. Yet that a great many epileptics cannot be relieved until this source of functional derangement is detected and remedied, I fully believe from results in treatment which in some instances have been too pronounced to admit of doubt. Among other questions, I always ask whether a bad breath is noticed, either as preceding the attacks or as occurring

during them. This symptom I find to be quite common, while in other cases constipation or diarrhœa is so associated, and not infrequently such clues have proved of the greatest value in directing the treatment.

The course of medical progress has been aptly likened to a spiral ascent, opinion repeatedly coming round toward former positions, but each time above, rather than at the level of the older views. The doctrines of cellular pathology and the great increase in the knowledge of the structural changes of disease, especially in the case of the heart, lungs, and kidneys, with their consequent aids to diagnosis, have displaced for years the humoral pathology of our predecessors. These advances have caused the stomach and bowels to lose much of the importance which they held in the minds of the old physicians as the chief centres of the processes both of life and of disease. Now, however, that chemistry is asserting her claims to be heard, as well as anatomy, the set of the current is once more in the old direction. It is curious, therefore, to find this tendency re-enforced even from the side of anatomy. It sounds strangely like a distant echo of the ancient teaching, that the origin of feelings, emotions, and moral characteristics is in the bowels, to find St. Paul, with his exhortation to put on "bowels of mercies," confirmed in a way by Dr. J. Bland Sutton, who says that "he is convinced that the spinal cord and brain of vertebrata have been evolved from what was originally a section of the alimentary canal; in other words, the central nervous system is a modified piece of bowel" (!).³ Dr. Gaskell also comes to the same conclusion by a different course of research, and states that "the tube which primarily represents the central nervous system in the vertebrate embryo must be regarded as a disused segment of the primitive alimentary canal."⁴

³ Brain, vol. x., p. 432.

⁴ Ibid., vol. xi., p. 336.

Periscope.

BY DRs. PETERSON, PRITCHARD, PICK, FISKE-BRYSON, LESZYNSKY
AND PECKHAM.

EXCERPTS FROM GERMAN JOURNALS.

BY FREDERICK PETERSON.

TUMORS IN THE REGION OF THE CORPORA QUADRIGEMINA.

Prof. H. Nothnagel writes upon this subject in the Wiener med. Presse, 1889, No. 3, basing his study upon ten cases collected by Bernhardt and four of his own. The first case described is as follows:

A boy, aged fifteen, fell from a tree, in 1885, and was unconscious and confined to bed for a short time. After a little he began to be unsteady in his gait and often fell to the floor. In the winter of 1886-1887 he suffered from severe headaches, pain in the eyes, nausea, and vomiting; later, optic atrophy, complete amaurosis, dizziness, and some deafness, the last due to chronic catarrh of the middle ear. Two symptoms especially were of value for diagnosis: first, a well-marked ataxia (Duchenne's *titubation cérébelleuse*); and secondly, the rigidity of the bulbi, whose movements were limited, particularly upward and to the left, more on the left than on the right side (there was paresis of the third and sixth nerves). The pupils were equal, their reaction sluggish. The diagnosis of tumor of the corpora quadrigemina with consecutive hydrocephalus was corroborated by autopsy. It was a papillary epithelial tumor, springing probably from the choroid plexus.

In the ten cases collected by Bernhardt was also one of his own, and since then he had observed three others.

Sensibility to light and vision, the reflex centre for the iris and the movements of the eye, some relation to general sensibility, blood pressure, vasomotor nerves, or corporeal equilibrium, all these had been at various times by various authors, located in the quadrigeminal bodies. Clinical features were often complicated by hydrocephalus, effects on distant or neighboring parts, etc. Hence, the author lays stress upon ataxia and paralysis of the ocular nerves as the chief factors in making a diagnosis. In almost all the cases there was the uncertain staggering gait. In three of his cases, all due to trauma, ataxia was the first symptom.

This could not be due to hydrocephalus, because the hydrocephalus only made itself evident sometime after the development of the ataxia. Pressure upon the cerebellum, or upon the crura cerebelli ad pontem did not seem to be a sufficient cause for the ataxia, since there were cases where the cerebellum was not affected, and yet ataxia was the initial symptom. This symptom seemed chiefly due to a disturbed function in the posterior quadrigeminal pair, since Gowers had described a case without ataxia in which only the anterior pair were affected. The ataxia is precisely like every other cerebral ataxia, such as that of drunkenness, and hence is not pathognomonic of quadrigeminal tumors.

The disturbances of vision, blindness, etc., depends chiefly upon choked discs, neuritis, optic atrophy, and hence nothing can be deduced from them as regards any direct relation to these bodies.

Kohts observed a boy with a posterior quadrigeminal tumor, who first presented a staggering gait, and only shortly before death slight visual disturbance. Paralysis of the ocular nerves was observed by Prof. Nothnagel in all of his cases; the bulbi were more or less immovable. The division of the paralysis was unequal, sometimes one part being more affected than another, or one eye more than another. In most of the cases, besides the third nerve, the fourth and sixth were also included in the injury. Such paralysis appearing in association with ataxia must lead one to think of a quadrigeminal tumor, since the nuclei are so closely aggregated in this region.

In one of the author's cases there was a tumor the size of a hazel nut, limited to the quadrigeminal bodies, including little injury to parts about the aqueduct of Sylvius, and leaving all other parts unaffected. This patient had no paralysis of ocular muscle, and presented a nystagmus only after several years. The nystagmus was probably due to slight irritation of the nuclei.

The localization of a tumor in the quadrigeminal region is to be based upon a combination of cerebral ataxia and simultaneous paralysis of ocular muscles on both sides in varying number and intensity. Hydrocephalus may also be associated. The diagnosis would remain the same even if, after the development of the above-named symptom, a hemiparesis, or hemianæsthesia should also be added, since the latter would merely be symptoms of pressure upon the pedunculus.

A CASE OF TUMOR OF THE PINEAL GLAND.

In the *Neurologisches Centralblatt*, May 15, 1889, Dr. Eugen Kny describes a case of tumor limited to the pineal gland, adding a ninth to the seven collected by Schulz (same periodical, 1886, No. 19), and the one reported by Daly ("Brain," July, 1887). The clinical history is briefly as follows:

J. H., aged thirty-two, suddenly began, in the spring of 1881, to have sharp pain in the occipital region, spreading in a year over both parietal and frontal regions. Since July, 1882, continual tinnitus aurium, and painful throbbing in the head. Later vertigo, blackness before the eyes, general tremor and periods of dimness of consciousness; gradually diminished vision. Feb. 1, 1883, choked discs, complete blindness in the right eye, light perceptible in the left.

August, 1884: Epileptiform convulsions. Dribbling of urine.

Nov., 1884: Nystagmus. Slowness of speech. Occipital pain continually.

May, 1885: Gradual weakening of the intelligence.

Nov., 1885: Strabismus divergens. Great tendency to fall backwards. No paresis. Anosmia. Cutaneous sensibility normal. In the last months of his life, dementia, soiling and wetting the bed. Wide rigid pupils. Bulbi peculiarly rigid, protruding, diverging. Epileptiform attacks.

Aug. 28, 1886: Death, with sudden sopor and rising of temperature to over 40° C.

Autopsy by von Recklinghausen: A lobular round-celled sarcoma of the pineal gland the size of a walnut. The corpora quadrigemina were not connected with the tumor, but flattened in front and pressed backwards.

PATHOLOGICAL FINDINGS IN A CASE OF TRAUMATIC NEUROSIS.

Drs. Sperling and Kronthal (*Neurolog. Centralb.*, June 1-15, 1889) give the clinical history and result of autopsy in a case of traumatic neurosis, which are condensed briefly as follows:

A man, aged forty-two, was so badly shaken up in a railway collision, in 1884, that he lost consciousness for a short time. A physician examined him and found bruises over his temple and abdomen. The patient complained only of a general feeling of fatigue. Within a few weeks a variety of inconstant symptoms appeared: there was psychic depression, apathy, irritability. He was easily startled by noises of passing trains, which would cause precordial pain

and a choking sensation. His sleep was harassed by frightful dreams. He had frontal headache, extending back to the neck and at times down the spine, formication in the hands or feet, ringing in the ears and flashes before the eyes. He had such a feeling of weakness that the slightest attempt at work caused general tremor. His gait was wide and so uncertain as to require the use of a cane. Musculature strong, and no disturbance of sensibility except the constant presence of painful points over the supraorbital regions. Knee-jerks and other reflexes considerably diminished. Sexual impotence. Palpitation.

A suit for damages was lost, physicians testifying that there were no objective symptoms, but probable simulation. Later, however, upon the testimony of Prof. Eulenburg and Dr. Sperling that the patient was entirely incapable of work, the decision was changed.

Hypnotism had a surprisingly good effect upon the patient. But January 27, 1889, patient died from cardiac and pulmonary disease.

The railway collision was not severe. No other person was injured, and none of the cars demolished.

The autopsy revealed a great degree of sclerosis, with here and there hyaline and fatty degeneration of the entire arterial system, but particularly in the cerebro-spinal vessels. There was a peculiar degeneration of the trunk of the sympathicus. In the spinal cord were scattered points of slight degeneration in all parts of the white substance, degeneration of the ganglia cells in a small part of the lower dorsal region, and a small hæmorrhage in the mid-dorsal region.

THE DIAGNOSTIC VALUE OF INCREASED KNEE-JERK AND ANKLE CLONUS.

Dr. T. Ziehen (*Corresp. Blätter des allg. ärztl. Vereins von Thüringen*, No. 1, 1889) puts in the form of a series of valuable clinical rules his experience with these reflexes:

Unilateral exaggeration of the knee-jerk is always significant of disease.

Bilateral exaggeration of the knee-jerk is only significant when ankle clonus co-exists.

Ankle clonus may be physiological in children; in adults it is pathological.

Ankle clonus and exaggerated knee-jerk occur in sixty per cent. of cases of epilepsy, while the plantar reflex is often strikingly weak.

The combination of diminished plantar reflex with in-

creased knee-jerks indicates functional, and not organic, disease.

Ankle clonus is found in twenty per cent. of cases of hysteria in general, but in a much higher per cent. of cases with hysterо-epilepsy, paralyses, anæsthesia, etc.

Ankle clonus is rare in paralysis agitans, very marked in paramyoclonus multiplex; exists in twenty to thirty per cent. of cases of neurasthenia; in some fifty per cent. of the psychoses; in tetanus, but not in tetany.

Exaggerated knee-jerks were found in 17.2 per cent. of criminals by Marro and Tombroso.

Exaggeration of deep reflexes in spastic spinal paralysis and amyotrophic lateral sclerosis differentiates decisively progressive muscular atrophy and neuritis, where they are absent.

Ankle clonus has the same relation to degeneration of the lateral columns of the cord as Westphal's symptom has to that of the posterior columns.

In ordinary apoplexy, with the loss of consciousness a primary foot clonus is observed, which shortly disappears. But if in two weeks to two months a secondary ankle clonus appears in the paralyzed leg, it means secondary degeneration of the lateral columns and leads to the certain anticipation of a permanent active hemicontracture.

Ankle clonus without other especial symptom leads one to suspect epilepsy or neurasthenia.

Ankle clonus with hemianæsthesia indicates hysteria; with intention-tremor, multiple sclerosis; with atrophy, amyotrophic lateral sclerosis; with spastic-paretic gait, spastic spinal paralysis or progressive paralysis; with anæsthesia of legs and paraplegia, dorsal or cervical myelitis.

Unilateral ankle clonus in hemiplegia or monoplegia indicates a cerebral lesion, and excludes almost always a spinal affection.

THEORY OF THE ORIGIN OF THE TRAUMATIC NEUROSES.

Th. Meynert discusses this subject in the Wiener klin. Woch., 1889, No. 24-26. He does not think there is any disturbance of the cortex, but that the functional disorder has its seat in the conducting tracts of the forebrain. The organic unilateral paralyses and anæsthesias are, as taught by Charcot, due to disturbances in the region of the arteriæ lenticulo-opticæ. Functional or traumatic hemiplegia is caused by nutritive disturbances in the region of the arteria chorioidea, which, according to Heubner and Duret,

nourishes the posterior segment of the internal capsule, the walls of the ventricles and their glia.

Kolisko's injection-experiments, not yet published, show that the nutritive region of the arteria chorioidea includes: the optic tract, internal capsule, wall of the descending horn and the cornu ammonis (smell centre, Zuckerkandl). Thus a nutritive disturbance of this vessel explains cerebral hemianæsthesia with unilateral blindness, anosmia and motor paralysis. The fact that the face and tongue are not affected in the functional disorders is explained by the position of their nerve tracts in the most anterior part of the posterior portion of the internal capsule, more on the border between the posterior and anterior limbs.

The anterior portion of the internal capsule receives its nutritive branches from vessels coming through the lamina perforata

The arteria chorioidea seems to have a predilection for functional disturbances, because it is the smallest branch of the basal vessels, and because it has no anastomoses with other basal arteries; hence, a vicarious distention or collateral overflow is not possible in its area when, for instance, there is a general spastic contraction of the circle of Willis. And in functional paralyses we are mostly concerned with spastic, vasomotor disturbances, such as irritation of the subcortical emotional centres and transfer of the irritation to the circle of Willis through the sympathetic.

EXPERIMENTAL OBSERVATIONS OF THE RELATION OF THE MOTOR GANGLION CELLS OF THE CORD TO THE PERIPHERAL NERVES.

Dr. Alb. von Sass, of Dorsat, contributes a paper upon the above subject to Virchow's Arch., vol. cxvi., part 2.

Prévost and David found in atrophy of the muscles of the hand atrophy of the roots of the seventh and eighth cervical nerves with atrophy of the anterior horn (especially affecting the cells of the lateral group) two to three cm. in length.

Sahli in a similar case found atrophy of the anterior horn at the level of the fourth to the seventh cervical nerves, particularly in the postero-external region.

Kahler and Pick examined the cord of a person whose arm had been amputated six years before, finding partial atrophy of the ganglion cells of the external group at the level of the fifth and sixth cervical nerves; while Hayem and Gilbert in a similar case found the motor cells in the whole cervical region affected, but more especially at the level of the seventh and eighth cervical and first dorsal segment.

Kahler and Pick located the centre for the calf muscles in the fourth and fifth lumbar segments; for the thigh, in the second to the sixth, but chiefly in the fifth and sixth (the middle group of cells).

Schultze placed the motor nuclei of the sciatic nerve in the lower portion of the lumbar enlargement, but the centres for the tibialis anticus and for the crural and obturator nerves higher.

Gudden, Mayser, Forel and others have induced atrophy of the nerve nuclei, experimentally, by section or removal of portions of peripheral nerves; and von Sass, making use of this method, has made the following localizations in the cord:

Median nerve—eighth and parts of the sixth and seventh cervical segments.

Radial nerve—Parts of the fifth and eighth and the whole seventh cervical segments.

Ulnar nerve—Upper half of the first dorsal, and the lowest and uppermost third of the eighth cervical segment.

Sciatic nerve—Lower half of the lumbar enlargement, and most strongly towards its middle.

The origin of the radial nerve was highest, generally speaking; that of the median, next; and of the ulnar, next; so that the muscles occupying the highest levels on the extremities seem to be represented in the highest levels of the cord.

HEMIATROPHY OF THE BRAIN.

At a meeting of the Budapest Medical Society, May 11, 1889 (*Centralb. f. Nervenheilk.*, June 15, 1889), Jacob Salgó presented a brain showing hemiatrophy. A young man of nineteen years, idiotic, had suffered from earliest childhood from epilepsy and left hemiplegia. The right hemisphere was hardly half as large as the left, and the convolutions, particularly in the occipital region, were as thin as paper. The change was least marked in the central gyri. The right basal ganglia were atrophied. The atrophy ceased at the pons; spinal cord normal.

Jendrassik remarked upon the case that the brain showed the lesion of a cortical hemiplegia. According to his observations upon such atrophic convolutions, the connective-tissue is increased and the nerve-fibres disappear. This pathological condition usually follows an acute infectious disease.

THE PSYCHIC AND SOMATIC DEGENERATION OF CRIMINALS.

Prof. Kirn contributes to the "Centralb. f. Nervenheilkunde," August 1, 1889, an interesting paper on criminal anthropology. He presents the views of the Italian school regarding the mental and physical abnormalities found in habitual criminals.

Among the primary *stigmata degenerationis* are those of the skull, such as microcephaly, makrocephaly, asymmetry, clincephaly, acro-oxy-cephaly, flat-head, and plagiocephaly.

Next in order to cranial changes are those of the face: the bird-face of the microcephalus, flattening of the upper part of the face in oxycephlus, deflection or flattening of the nose, protrusion of the superciliary ridges, asymmetry of the orbits, inequality of the position of the teeth, malformation of the lips and palate, progænia, and finally the numerous abnormalities of the ear.

The rest of the body may present peculiarities such as dwarfishness, giant growth, asymmetry of the two halves of the thorax, club-foot and club-hand, and various anomalies of the sexual organs.

Among certain common functional disturbances are diminution in sensibility, particularly analgesia, color-blindness, lack of power to blush, left-handedness, and abnormal sexual instinct.

To the series of psychic anomalies belong weak intellect, lack of uniformity in mental development, a want of sufficient comprehension of the immorality of crime and consequent impossibility of remorse or improvement, emotional inconstancy, and a tendency from childhood to evil and wickedness. From these facts several important conclusions have been drawn:

1. Congenital criminality is identical with moral insanity. Both conditions exhibit the same physical defects and malformations, and the same mental deviations; and both are usually congenital, becoming worse at puberty.

2. Epileptics are nearly related to criminals, for analogous bodily and mental stigmata are common to both.

Regarding the above teachings of Lombroso and his followers Prof. Kirn expresses his own beliefs as the result of his experience.

He says the symptoms of somatic and psychic degeneration that have been described, although found in a large number of habitual criminals, are not by any means generally present, and have therefore only a relative value.

But experience in court and prison certainly shows that many deeds punished as crimes are committed under the influence of certain anomalies of brain function. The most important of these is weak-mindedness, which may be either congenital or acquired. In the former case there is defect or malformation of the brain; in the latter there is psychic degeneration due to alcoholism, epilepsy, injuries to the head, or chronic cerebral disease. There is a pathological inferiority or perversity; some habitual criminals remain on a low plane of mental development, others exhibit a pathological growth.

Prof. Kirm thinks that great weight should be laid upon cranial anomalies, since they are in fact sometimes observed in habitual criminals. But there is no characteristic defect or shape of the skull that may be regarded as pathognomonic.

Extensive craniometrical study has shown that there is in many of this class a tendency to abnormally small, and and in some to abnormally large, skulls; but from this one is permitted to conclude only that a certain disposition exists in a number of criminals to morbid cranial and encephalic development.

The structural anomalies thus far described, in the brains of criminals, are too few and not sufficiently corroborated to allow decided conclusions to be drawn from them.

As regards the psychic symptom-complex, no uniform criminal type can be portrayed, but only various mental anomalies which exist in widely differing combinations in this class.

He does not believe in the identification of congenital criminality and moral insanity. The latter is not an independent disease, but only a symptom-complex, sometimes congenital in connection with other signs of defective development, sometimes acquired in conjunction with epileptic, paralytic, alcoholic and traumatic psychoses. Moral insanity, which may be observed in the most heterogeneous psychoses, may under conditions also make its appearance in the criminal nature. In opposition to the views of the Italian school, he thinks there is a total want of analogy between the epileptic neurosis and criminality.

He finally concludes that an anthropological criminal type has decidedly no existence, but that there are to be found in prisons not a few individuals who present marked somatic defects and malformations, defective mental development, or psychic degeneration due to disease.

AN INSTRUMENT FOR THE PERCEPTION OF LIGHT THROUGH
THE SENSES OF TEMPERATURE AND POSITION.

Dr. Noischewski presented to the Neurological Section of the Society of Russian Physicians, in January, 1889, an instrument which he calls the electrophthalmocyclop. It consists of a small camera obscura, whose posterior wall is composed of three layers. The first is of fine metallic gauze, the second a closely approximated plate of selenium, and the last a brush-like arrangement of gold wires which are to come in contact with the skin. This veritable cyclop is applied to the middle of the forehead, and the light-rays falling into the apparatus are metamorphosed into a thermoelectric current, which is at once perceived by the glabellar nerve filaments, for the forehead has the senses of temperature and position particularly well developed.

The inventor draws the following conclusions from his experiments with the instrument :

The presence of a light-giving or of an illuminated object is manifested in the perceptive field as a sensation of warmth.

A light object on a dark background is perceived as a peripherally warm sensation, with a sensationless centre.

The degree of the sensation of warmth increases with the approach of the illuminating object, and *vice versa*. A movement of the feeling of warmth toward the right shows that the light has moved to the left, *vice versa*.

If the warm area moves downwards, the illuminating object is moving upwards, and *vice versa*.

Hence the blind will be able to see light through the mediation of tactile sensibility. It is possible for them by means of this apparatus to perceive the presence of a light object, its position and its movements from side to side, up or down or nearer and farther away.—*Centralb. f. Nervenheilk.*, March, 1889.

F. P.

EXCERPTS FROM THE RUSSIAN.

By Drs. PRITCHARD AND PICK.

ON THE PHENOMENA APPEARING IN ANIMALS AFTER
SECTION OF THE POSTERIOR COLUMNS OF THE SPINAL
CORD, AND THE RELATION OF THE LATTER TO THE
EQUILIBRIUM OF THE BODY.

Prof. W. Bechterew (*Wjestnik psichiatrii i nevropatologii*, 1889, vii., 1). The writer reports upon a series of experiments on doves, rabbits, and dogs, in which he sev-

ered the posterior columns of the spinal cord. The section was made at the cervical portion, preferably in its upper portion. In a few experiments on dogs the section was confined solely to the columns of Goll; in some the nuclei funic. grac. were only severed. Observation of the animals extended over a period of from some days to several months. The extent of the experimental procedure was controlled by postmortem and sometimes by microscopic examination.

At the moment of severing the posterior columns and immediately after, the animals show reflex movements of general unquiet; yet these soon disappear, and then one observes, as a constant effect of the operation, disturbances of movement, which may be interpreted as disturbances of equilibrium. Neither paralysis nor paresis of the extremities makes its appearance; the animals retain their capability of locomotion; they can walk, stand, run; co-ordination of single movements remains uninfluenced: yet, in standing as well as in running, a remarkable insecurity is observed; the animals tumble either forward or backward, or to either side. The flight of the doves operated upon is slow and difficult, and their bodies assume then a peculiar shape; the jumps of the rabbits operated upon are irregular; the dogs walk with their legs spread far apart, and their bodies sway from side to side. Blindfolding the eyes causes in all cases a greater prominence of these phenomena. If the animals remain alive a longer time, then a gradual decrease and sometimes, after several weeks, complete disappearance of the disturbances could be seen.

As to the sensibility, the writer found it in no case to be reduced; the muscular sense of the extremities was also not disturbed. On the contrary, anæsthesia of the forepaws, together with loss of the muscular sense and disturbance of co-ordination, could be seen, in case the section was made at the height of the fifth to the sixth cervical vertebræ.

In some cases the writer observed, in his animals experimented on, long-continuing hyperæsthesia, and indeed when section was made at the place of lesion it revealed inflammatory changes of the gray substance. The above-described disturbances of equilibrium made their appearance in the same way in dogs, in which not the entire column, but that of Goll was severed or the nuclei funic. grac. injured.

The writer concludes from his experiments that the posterior columns are a path of conduction, which inter-

ruption causes a disturbance of the equilibrium, with retention of sensibility.

A CASE OF SEMI-LATERAL FACIAL ATROPHY.

L. Blumenau (*Wjestnik psichiatrii i nevropatologii*, 1889, vii., 1). A factory operative, fifty years of age, presented distinct atrophy of the right lower half of the face; the cheek was here fallen in, the skin wrinkled, the lower lip thin, and the nostril narrower than on the opposite side. The atrophy also extended to the right half of the tongue, the soft palate, and the maxillary bone. His moustache only grew upon the left side; the right side of the upper lip was completely hairless. The frontal region of the face was not affected. The sensibility of the skin, electric excitability, and peripheric temperature were normal on both sides of the face.

The development of the morbid process began in his sixteenth year; the patient observed then a whitish spot upon the upper lip. In his course of five years the atrophy reached its present extent, and since then has remained without change. No circumstance could be found in his history to which one might attribute ætiological importance.

COMPRESSION OF THE SPINAL CORD IN CONSEQUENCE OF FRACTURE OF THE SECOND DORSAL VERTEBRA.

J. Anfimov (*Wjestnik psichiatrii i nevropatologii*, 1889, ii.). A man, thirty-four years of age, and up to that time in good health, employed upon a street-railroad. July 28, 1888, was run over by a droschky, by which he got a violent blow between the shoulders; he fell prostrate and was thrown to one side. He did not lose consciousness and was able to raise himself and walk home, about fifteen kilom. away, where, about two to three hours afterward, sudden and complete paralysis of the lower extremities made its appearance. He was brought at once into the surgical clinic, where, besides complete paraplegia, inferior, paralysis of the bladder and rectum was diagnosed. Loss of sensibility over the entire body below the second rib was also remarked; temperature $37^{\circ}\text{C}.$, pulse 48 in 1; painfulness to pressure of the cervical vertebræ. The next day an attack of asphyxia appeared, which was removed by artificial respiration and injection of ammonia; in the days following, phenomena of fever and decubitus. September 13th he was received into Prof. Mierzejewski's clinic in the following condition:

Complete loss of cutaneous sensibility and of the muscular sense over the entire body below the second rib and the upper dorsal vertebræ; complete flaccid paraplegia inferior; paralysis vesicæ et recti; complete absence of the tendon and skin reflexes of the lower extremities and trunk; loss of the mechanical muscle; excitability and great reduction of the electric; paralysis of the intercostal muscles and difficult respiration, chiefly affecting the diaphragm and the cervical muscles; the movements of the head, upper extremities, and further the consciousness and speech undisturbed; extensive decubitus in several spots, with gradual aggravation of the general condition and increase of the dyspnœa. Death occurred September 18th. Two days before, somnolence had added itself, together with acceleration of the pulse and high temperature, to the list of symptoms.

Post-mortem examination revealed an oblique fracture of the second dorsal vertebra, with splintering of the bone, dislocation of the cartilage, and hæmorrhage in its surroundings. Corresponding to this place, there was found on the spinal cord, at the place between the cervical and the dorsal portions, a focus of softening, about one inch in length, in the whole breadth of the substance of the spinal cord. The softened place was reddish colored; the dura mater in the neighborhood of the fractured vertebræ was thickened, hyperæmic, and adherent to a splinter of bone, which had been broken off. Below the softened place the appearance of the cord was normal. Microscopic investigation of the same revealed ascending and descending degeneration; otherwise the tissue of the cord, even in distant regions—as, for example, from the lumbar enlargement—seemed dull, yet without distinct pathological changes.

In considering the case, the writer calls attention to the difficulty of explaining the absence of 'the tendon-skin reflexes in compression of the cord.

A similar case was published, a short time ago, by Leyden and Jürgens (Berliner klin. Wochenschr., 1888, Nos. 22 and 24). In this case they would ascribe the absence to the trauma being so violent as to cause an irritation of the cord and subsequent inhibition of the reflexes.

P. & P.

ERB'S PARALYSIS.

The "Lancet" (March 1, 1890) records two cases that were exhibited before the Medical Society of London. The first was a man, thirty-one years old, who fell down-stairs, after drinking too much, and struck the point of the shoul-

der, causing inability to use the joint. The deltoid, supra-spinatus, infra-spinatus, pectoralis major (slightly), triceps, brachialis anticus, and supinator longus were affected, reacting only slightly to faradism, the deltoid not at all. The faradic current was slowly bringing about a cure.

The second case, a man of thirty-seven, was pitched from a cab, falling on the left shoulder and side of the head, stretching and straining the neck. He gradually lost the use of the arm. About the shoulder there was some anæsthesia at first. The biceps, coraco-brachialis, brachialis anticus, deltoid, and supra-spinatus were completely paralyzed. To strong faradism there was no reaction, the muscles giving the reaction of degeneration. A stretching of the brachial plexus is the cause of nearly all such paralyses, this stretching corresponding to a lesion of the fifth and sixth cervical nerves before joining the plexus. In the monkey, paralysis of the biceps, supinator longus, and deltoid follows the division of these nerves.

A woman, falling asleep with the neck resting against the edge of the table, found herself in this same unhappy plight on awaking. The same thing might happen in infantile paralysis; without anæsthesia, however, the lesion being situated in the anterior horns.

In answer to questions, Dr. Beevor, who exhibited the last case, said there had been no pupillary phenomena, which could only occur if the second dorsal nerve was also affected. He believed that the supinator longus was not a pronator or a supinator, but a pure flexor, and that the action of a muscle produced by faradism was not the same as that produced by the will.

MALARIAL NEURITIS AND NEURO-RETINITIS.

The "British Medical Journal" (March 9, 1890) has a paper on this subject, by N. C. Macnamara. In places where malaria prevails, says the author, hemicrania, sciatica, and loss of sight, in connection with intermittent fever, are not unfrequently met with. A case cited, a former tea-planter in Assam, had frequent attacks of intermittent fever; and several times, accompanying such attacks, impairment of vision that prevented reading and writing for ten days or a fortnight. An attack of ague in England produced in a few hours marked disturbances of vision. With the right eye he could only decipher the letters of Snellen 2.25; and with the left fingers could only be dimly counted. The pupils acted imperfectly to light, and were somewhat dilated. There was no pain, photophobia, or

conjunctival congestion. The optic discs were completely obscured by effusion, which extended into the retina. The retinal veins were tortuous and congested. There was no albumen, no sugar, in the urine. Under quinine, then arsenic and strychnine and a change of air, the discs gradually cleared up. Two months elapsed, however, before the patient could read Snellen 1.25. In six months the optic discs were white; Snellen 0.5 could be read; vision in the right eye was $\frac{6}{8}$, in the left $\frac{6}{9}$; and there was no return of fever.

In another instance, where there was complete blindness—but no suspicion of syphilis, no albumen, no rheumatism—full doses of strychnine and a bracing climate restored sight in about a year. A boy of ten recovered under similar treatment.

Another case added to almost total blindness the annoyances attendant upon paralysis of the left ulnar nerve, together with great difficulty in swallowing. Within three months, under the anti-malarial treatment referred to above, complete power over the nerves and muscles whose functions had been lost was restored. In none of the cases cited was anæmia present, though enlargement of the spleen existed in every one. Kidney trouble, syphilis, and rheumatism were excluded. Impaired vision remained for a long time the only malarial symptom. Except for it, the patients were practically well, unless exposed to sudden cold or damp. Checking the fever prevented the probable atrophy of the disc.

HEREDITY AS A FACTOR IN ALCOHOLISM.

In Paul Sollier's treatise on this subject ("Prix Aubanel," 1889) the following generalities are given as rational conclusions derived from the careful and discriminating study of some 350 families who have numbered one or more idiotic, epileptic, or mentally unsound representatives in the wards of the Bicêtre

I. There exists a form of passion for drink that finds its true place of classification somewhere between dipsomania, hereditary insanity, and acquired alcoholism. This is hereditary alcoholism, more frequent in occurrence than dipsomania and having much in common with acquired alcoholism.

II. Hereditary alcoholism may be identical with its source, or different in its manifestations, the proportion of the first to the second being as three to four.

III. Hereditary alcoholism belongs to neuropathic humanity, particularly to its psychopathic division.

IV. Causes that produce hereditary alcoholism, especially among the descendants of drinkers, are not occasional or apparent. The seeming and chance causes that produce such direful results are by no means the important factors we have been led to believe. The only true cause is that *heredity* creates the predisposition, the desire, together with the intellectual and moral state that renders the individual powerless to resist.

HOW WHOOPING-COUGH IS TREATED IN SPAIN.

"L'Union Médicale" (March 6, 1890) gives the following, from the report of the Scientific Congress of Barcelona: Carreras advises in whooping-cough an application of resorcin to the pharynx and to the vestibule of the larynx. He also gives large doses of chloral to allay the excitability of the superior laryngeal nerves. Guerra y Estapé also approves of resorcin locally, and when the cough becomes non-convulsive finds benefit from balsams. Calatreveno employs belladonna, inhalations of benzoate of soda, and insufflations of powdered roasted coffee and sulphate of quinine mixed. He also gives doses of antipyrin, one-hundredth of a grain for every month the patient has lived.

L. F. B.

A CASE OF ALEXIA (DYSANAGNOSIA).

In the January number of the "Archives of Ophthalmology," Dr. Swan M. Burnett, of Washington, reports the history of the following unique case: The patient was a clergyman, eighty-two years of age. Some weeks before, while assisting his servant in watering the grass, he staggered, but did not fall, and, feeling uncomfortable, went into the house. He passed a comfortable night, and came down-stairs the next morning as usual, read the service, and had the customary family devotions. Soon afterward, however, he complained of feeling bad, was taken up-stairs, and almost directly went into strong general convulsions. Of these he had three during the afternoon and evening, and afterward passed into a condition of stupor, from which he could be roused with difficulty and for only a moment. At the end of three days he became conscious, but was very weak. On the fourth day, in the evening, he read some from the prayer-book, but was very much fatigued thereby. On the next morning he attempted to read again, but found he could not.

On examination he failed to name any letter of Snellen's test-type correctly, even the largest. The refracting media were unusually clear for a person of that age, and the fundus

of the eye did not present anything sufficiently abnormal to account for such a marked deterioration of vision as seemed to be present.

It was not that he could not see the individual letters of the word, or the word itself, but that they failed to convey to him the same ideas they had for the last seventy or seventy-five years. He was unable to read anything correctly. A word here and there might be properly called, but the sense of even the shortest sentence would be ridiculously misinterpreted.

For example: The morning paper was lying on the table, and he was asked to read aloud: "Judge Thurman will formally open his campaign at Port Huron to-day." This he read as follows: "John, John then the hatter his hat going to be h—green."

No. 12, of Jäger, reads thus: "The keys and he began playing a sad and infinitely lovely movement." His interpretation of it was: "Was told to be and haying a a was to be be ing in mo on when he was crydt."

Even these attempts at reading were made slowly and hesitatingly, and often with the necessity of an effort to decipher a word by spelling, just like a child learning to read.

Having been a close student and constant reader all his life, he was himself aware of this being the veriest nonsense.

His trouble is confined entirely to an inability to interpret the meaning of printed or written words by means of the impressions they make on the retina. When anything is read to him he understands it perfectly, and can repeat it accurately; and his memory of things he has read before is unimpaired, and all other impressions made on his retina are properly interpreted.

He can even read numbers correctly, and can tell the amount of a check, though unable to tell to whom it is drawn or by whom.

And not only are the Arabic numerals recognized without difficulty, but he is able to interpret correctly the Roman numerals also. All the letters he can distinguish individually with ease except the letter "s." To look at this is always disagreeable. The word "the" is seldom or never misinterpreted. All kinds of pictures he understands and enjoys. He can write either originally or from dictation, but is as unable to read his own writing as he is that of any one else or printed matter. It is necessary, however, that he write continuously. If interrupted, he cannot go back and begin where he left off. Yet it is possible for him to

break a line in writing if he is not interrupted in his own line of thought.

His memory for what he has read before is not impaired.

The notes in this case were made nearly a year ago, and the patient succumbed to an attack of pneumonia on January 20, 1890. There was no autopsy. His general mental faculties remained unusually clear and bright to the last, and his bodily vigor was as it had been for many years past until seized with the prevailing influenza.

The author agrees with Nieden in the opinion that the term "dysanagnosia" is etymologically more correct and scientifically preferable to either "alexia" or "dyslexia," the two latter being hybrids of Latin and Greek, whose adoption in our nomenclature should not be encouraged.

W. M. L.

FRONTO-FACIAL ASYMMETRY OF THE INSANE.

Dr. R. Roscioli (*Il Manico*, 1889, v., p. 27), having in mind the earlier works of Lasègue, Amadei, Venturi, etc., on the importance of an asymmetric formation of the frontal bone and the facial cranium, examined most carefully 388 insane persons of the most various shapes, etc., and 100 sane persons. Entirely symmetric skulls he only found in about thirty per cent. of the insane and in sixteen per cent. of the sane subjects.

He does not ascribe any value to asymmetry of a slight degree, not even for the showing of a predisposition to insanity. The higher degrees of obliqueness of the skull are found in epileptics, but also in those suffering from other mental diseases, and in sane persons also; they, however, are to be regarded as a sign of degeneration, and are the more frequent the more distinct the group of diseases in question are to be regarded as a degenerative psychosis. Striking asymmetry would be able to essentially obscure the prognosis of a case of mania or melancholia progressing apparently favorably. Sommer, in his examination of skulls of the insane, has nearly always found cranial asymmetry present (*Virchow's Archiv*, Bd. 89 und 90); only 3.5 per cent. were symmetric. In agreement with Zuckerkandl, Meynert, Dohrn, and others, he thinks persistent asymmetry may not rarely be traced back to mechanical shifting of the cranial bones (*intra-partum*). 3.5 per cent. asymmetric skulls corresponded to the 3.6 per cent. breech-presentations, etc., while the first cranial position corresponds to a flattening of the left parietal and frontal region, and the

second cranial position, *vice versa*, corresponding to the flattening of right parietal and frontal region in nearly the same frequency, usually 1 : 2.7 to 1 : 2.3. Those asymmetries dependent on presentations of the ends of the cranium will be the more pronounced the greater the misrelation between the internal pelvis and the volume of the head. A narrow pelvis in the mother, a striking difference between the length of the large father's and the small mother's head will bring an influence to bear upon the child's head, which through these influences becomes relatively or absolutely too large, which cannot be otherwise than unfavorable. Especially is this the case in the crania of those children born of weakly parents, where orchitis, cerebral hypertrophy, etc., add to its size. (The crania of the insane are known to be more capacious than those of the sane.) Perhaps sometimes the so-called hereditary predisposition to insanity may be traced back to some such individual peculiarity, and indeed to some traumatic crushing and shifting of the brain during labor.

G. P.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, Tuesday Evening, March 4, 1890.

The PRESIDENT, DR. GEO. W. JACOBY, in the chair.

Dr. HENRY D. NOYES and Dr. C. L. DANA reported a case of

TUMOR OF THE MEDULLA AND LEFT ANOPHTHALMUS.

A. K., aged thirty-seven, had a severe headache for nearly a month, some seventeen years ago, followed by dimness of vision in the left eye. The centre of the visual field appeared dark and became almost blind in that eye in 1875. A large cysticercus cellulosæ was then removed with this eye. He had no further trouble until February, 1888, when there was a recurrence of a similar headache in the same part of the head, on the left side at first, later general. Other symptoms then began to manifest themselves: vertigo, tinnitus aurium, numbness and weakness and exaggerated reflexes upon the right side, paresis of the right

internal rectus, staggering to the right side, ptosis, nystagmus. Later, in January, 1889, there was paresis of both externi, nystagmus more marked, weakness of left masseter and atrophy of left temporal muscle.

February 11th: Anosmia. Taste very much blunted. The next day epileptiform convulsions.

February 13th: Operation for traumatic stricture.

February 16, 1889: Death.

The autopsy showed three tumors in the floor of the fourth ventricle exerting pressure upon the left middle cerebellar peduncle, and slight pachymeningitis interna fibrosa. The left optic nerve was very much smaller than the right. The optic tracts were very nearly equal in size, but the left was a trifle larger than the right. The external geniculate body could hardly be made out upon the left side, and that on the right seemed smaller than normal. The left ant. corp. quad. seemed smaller than the right.

Each of the three tumors measured some 16 mm. in diameter. One was on the right and two on the left of the median line. The one on the right was more anteriorly situated, extending from the middle of the pons to the post. corp. quad. of the two on the left side, one extended from near the calamus to about the edge of the pons, and was intimately connected with the third tumor. This last extended into the left half of the pons and middle cerebellar peduncle. They were fairly well defined, but somewhat infiltrating sarcomata.

The occipital lobes were of nearly equal size. A careful microscopical examination had been made and the details were reported.

There was compression and more or less destruction of the following nervous structures:

The left lemniscus, left pyramidal tract, left eighth, ninth, and tenth nerves and nuclei, left and perhaps right third nerves—probably the fourth, sixth, and seventh nerves, though these could not be examined.

Among the more important conclusions drawn by the authors were the following:

The oldest and most extensive process was on the left side.

The right hemiparesis was due to pressure on the motor tract in the pons and medulla. The external ocular muscles were involved without interference with the ciliary muscle and iris, and in an order which confirmed the arrangement of the oculo-motor nuclei, as given by Hensen and Völcker, rather than that of Kahler and Pick, or of Starr.

Furthermore, the case showed apparently a total decussation of the optic-nerve fibres.

Dr. SACHS said that the unusual features of the case rendered discussion difficult. There was a twofold morbid condition, and it was not easy to determine where the one process began and the other ended. The symptoms were much more complicated than in ordinary affections of the medulla oblongata, as illustrated by a case at present under his own observation, in which there was a syphilitic tumor of the medulla giving rise to hemiatrophy of the tongue and a paresis of all four extremities, from which the patient was now recovering. He did not feel convinced that the ocular symptoms in the case of Drs. Noyes and Dana were of nuclear origin. He believed that symptoms of supposed nuclear origin were at times found to be due to the involvement of the intra-cerebral root fibres. It was unfortunate that this specimen was not hardened sufficiently well to permit of exact determination of the parts destroyed in this case.

Dr. E. D. FISHER read a paper upon the

PATHOLOGY OF CHOREA.

Dr. GRAY did not agree with the author's idea of the pathological processes in chorea. He thought the results of Dr. Dana's recent pathological researches in this disease much nearer the truth than the facts cited by Dr. Fisher. Nor could he quite coincide with the latter's view that chorea could not be relieved by peripheral measures. He had known of cases of chorea to recover in a few days by the mere change of residence from the city into the country, which was a salient example of the benefit of peripheral changes. Some years ago, when circumcision was in vogue, he had tried it in cases of chorea with considerable improvement. Dr. Mitchell had shown that there was a relation between chorea and barometric disturbance, and he had himself noted that in ante-cyclone periods cases of chorea were worse, and some of the worst cases would break out.

Dr. DANA believed that no one as yet knew what is the pathological anatomy of chorea, although the truth is nearer than it has been before. He considered the disease to be a species of fright-neurosis, affecting vaso-motor centres and causing hyperæmia of the brain. In Sydenham's chorea there seemed to be almost a paralysis of the vessels at the base of the brain. The disorder was so often due to emotional causes that it might be classed with the traumatic

neuroses. Sometimes, of course, there was a humoral element in its etiology, such as gout or rheumatism. He thought Dr. Fisher's ideas applicable to a certain class of cases, though we should distinguish between Sydenham's and Huntington's choreas, post-hemiplegic chorea, tic, etc.

Dr. SACHS desired to ask some of the ophthalmologists present as to the relations of habit chorea to ocular and nasal troubles, upon which so much stress had been laid. He believed himself in the treatment of these forms of chorea as ordinary cases, only that they are limited especially to the face.

Dr. WEBSTER had had considerable experience with such cases, but it was impossible to tell what proportion had been relieved by ocular treatment. The majority of his cases had been of spasmodic nictitation, sometimes extending down the side of the nose and even to the lip. Treatment with atropine for several weeks frequently relieved them. Sometimes glasses were used in addition. All such cases should have any ocular defects corrected.

Dr. NOYES felt very skeptical as to the value of the methods which had been suggested for the relief of these spasmodic affections of the face. Dr. Sachs probably did not refer so much to nictitation, which was often, although not always, due to ocular irritation as to facial tic, which was a very obstinate disorder and in which ocular treatment was not particularly valuable. A very marked case of this kind was that of one of the physicians in the city. Some astigmatism and muscular insufficiency were corrected, and though considerably relieved for several years, he was not cured. Three weeks ago he returned for further treatment, but he was unable to help him. Many years ago he used to cut the facial nerve to afford temporary relief. In chorea he thought the correction of eye troubles only subordinate to general hygienic and medical treatment.

Dr. KNAPP had also treated cases of chorea and facial spasm, some of them with great myopia and insufficiency of ocular muscles. He detailed two cases that had been much improved by such treatment. Other cases had shown scarcely any relief. He had known of a few cases operated upon several times, and whereas the eyes were formerly very good they were now incapacitated for use, and the facial spasm was no better.

Dr. THOMSON said he was a decided humoralist as regarded the pathology of chorea. Even cases supposed to be due to shock and fright would be found on careful

examination to be in reality caused by a rheumatic condition. Rheumatism does not invariably show itself in articular or muscular symptoms, and he had observed many cases without these and yet with cardiac manifestations. When one considered the undoubted relation between nervous and humoral states,—how gout, for instance, might suddenly disappear by fright—one could not feel like adopting a great many causes for one definite group of facts. There was no reason why the rheumatic poison might not at one period of life manifest itself by cardiac symptoms and at another by articular. In the chorea of children he had frequently discovered endocarditis, or a rheumatic element of some kind in them or in their relatives, and he thought the poison had a selective affinity for the cerebral cortex. He would treat all cases of chorea, even those with local manifestations only, as of humoral origin. The poison might produce sclerotic changes in the brain possibly, as Dr. Fisher had suggested.

Dr. FISHER, in closing the discussion, said he agreed with Drs. Noyes and Knapp that ocular defects should in any case be removed, although in the majority of cases no direct effect could be positively ascribable to the operation.

He could scarcely agree with Dr. Thomson's statement as to the predominating influence of rheumatism as inducing chorea. Dr. Thomson's explanation of the pathological conditions present in chronic chorea as consisting of degenerative changes in the cortex accorded with his own remarks in the body of the paper. He was disposed to believe with Dr. Gray in the good results of change of air and of removal of local irritation in acute cases, but in chronic chorea such means, while improving the general nutrition, did not cure the disease.

The following nominations were made for the ensuing year:

For President—Dr. Landon Carter Gray.

“ First Vice-President—Dr. B. Sachs.

“ Second “ “ —Dr. E. D. Fisher.

“ Recording Secretary—Dr. Frederick Peterson.

“ Corresponding “ —Dr. W. M. Leszynsky.

“ Treasurer—Dr. Græme M. Hammond.

“ Councillors—Drs. G. W. Jacoby, C. L. Dana, M. D. Field, M. Allen Starr, J. C. Shaw, and E. C. Seguin.

FREDERICK PETERSON,

Recording Secretary.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, February 24, 1890.

The President, Dr. H. C. WOOD, in the chair.

Dr. J. CHALMERS DA COSTA read a paper on "Ophthalmic Migraine." (See page 217.)

DISCUSSION.

Dr. H. C. WOOD stated that he had recently been consulted, by a professor in one of the Western colleges, by letter. The patient was thirty-five years old, and had suffered occasionally from attacks, similar to those now present, between fourteen and twenty years of age, the attacks always following extra exertion. From 1876 there had been no attacks until the latter part of 1889. The professor describes these paroxysms as follows:

"The attack begins by imperfect vision; usually there appears a spot in the field of vision. Sometimes this spot appears to dance, at other times it simply obscures a portion of the object seen. If I run the eye along a printed line, as in reading, it appears exactly as if something were being drawn along the line, covering up the words as fast as I pass them. If I attempt to call the words from right to left, I cannot do so rapidly, but must wait for the curtain, as it were, to be drawn aside. This feature of the attack, which I shall designate the 'blind spot' stage, usually lasts twenty or thirty minutes. This is always present. Other features of the attack are sometimes wanting. There is usually a sensation of numbness at some time during the attack. Occasionally it precedes the 'blind spot' stage, but often comes on after the latter has disappeared. Sometimes both features are present at the same time. The numbness is usually in hands and fingers, sometimes in forearm. It is frequently in cheeks and lips and tongue. This numbness is not like that resulting from cold, neither like that from partial paralysis of a member, called 'being asleep;' it resembles the latter more than the former. When this numbness is in face or tongue, there is always a dancing or twitching sensation. This feature varies as to its time of appearance and as to its duration.

"Another feature is the severe pain in the head, or rather in the eye. The pain seems to be in the region of the right eye. This pain does not begin until about the time the 'blind spot' stage disappears. The pain lasts some three or four hours. I feel quite well then until the next attack. It may be well to say that I now take five grains of antipyrine when I feel the attack coming on, and

thus avoid much of the pain in the eye. Twice, in the last ten attacks, I did not take the antipyrine immediately, and the severe pain in the eye on those two occasions shows that the almost absence of pain on the other eight occasions was due to its use. As well as I can remember, I have had about fifteen of these attacks within the last four or five weeks."

In subsequent letter Professor ——— stated that he had discovered that his mother had suffered from attacks precisely similar to his own, even to the numbness and imperfection of vision.

There are one or two points worthy of note in regard to these cases. In the first place, as to the theories suggested to account for the disturbance. I think that at present any one has a right to believe any theory he chooses, for all our theories are houses built of cards, as we have not sufficient data to enable us to formulate a positive theory. We can simply say that megrim is of the nature of a nerve-storm, and that this nerve-storm sometimes involves various sensory areas, and more rarely even the motor areas.

I think that in this country severe ophthalmic disturbances are infrequent. I can remember but one pronounced case of ophthalmic megrim; possibly, when there is much disturbance of vision, the cases go to the oculist, rather than to the neurologist.

Many years ago Trousseau alleged that there is a relationship between megrim and epilepsy. I have seen many cases of epilepsy and many cases of megrim, but I have never yet seen a family in which megrim alternated in successive generations with epilepsy. I have seen only one case of megrim in an epileptic, and in that instance there seemed to be no relation between the paroxysms of epilepsy and those of megrim. There may, however, be a form of megrim which is epileptic megrim, just as you may have epileptic gastralgia and epileptic sensory discharges in other parts of the body, replacing the ordinary epileptic motor discharges. But the rare occurrence of such cases is no more proof of relationship between epilepsy and ordinary megrim than is the occurrence of epileptic gastralgia evidence of a relation between epilepsy and ordinary stomach-ache.

Dr. JAMES HENDRIE LLOYD.—I know very well a patient who has been the subject of ophthalmic megrim since childhood. The symptoms are similar to those referred to by Dr. Wood's case. The vision becomes blurred and indistinct, the idea of a curtain before a part of the field of vision

being rather a happy expression of this. This passes off in ten or fifteen minutes, and is replaced by severe headache, unless, before that develops, the patient obtains relief by saline cathartics. I have associated the occurrence of these attacks with gastric disturbance, although usually the patient is not sick at the stomach until after the storm has taken place. These attacks recur periodically two or three times a year. In regard to the hereditary nature of this affection, I believe that one of the patient's grandfathers suffered with it.

Dr. J. MADISON TAYLOR.—I too might add a word of personal experience. Myself and a large number of my relatives suffer from what seems a true migraine—headaches of regularly periodical recurrence, and seemingly unrelated to accidental causes, always excepting worry or nervous strain. These begin in childhood, reach a climax of severity in early adult life, and then happily lessen, both in frequency and severity, till they sometimes cease.

In certain instances it is strictly one-sided, and often localizable in one spot. It is almost always accompanied by increased pain on using the eyes, but no marked visual disturbances. It is interesting to note and satisfactory for me to report that neither in the score or so of cases thus reviewed, nor in the remainder of a pretty large connection, is there any instance of true nervous disorder, except here and there a little neuralgia, nor indeed of any of the diathetic diseases.

The paroxysms are accompanied by coldness of the extremities, with seemingly increased heat of head. The one drug which most efficiently relieves, as I have discovered after much search, is atropia, especially used hypodermatically. The instant the peripheral arterioles begin to relax their spasm, under the benign influence of this rapidly acting drug, the heart quiets down, pain lessens, and, along with this, the often more distressing nervous depression and restlessness.

I hazard the conjecture that, whatever be the *fons et origo* of this disorder, it is usually complicated by a certain amount of intestinal disturbance and fæcal reabsorption. The phenomena of aparoxyism closely resembles ptomaine poisoning, and the treatment suitable for each is much the same.

Dr. H. C. WOOD.—I have noticed in at least one case that during a paroxysm of megrim the jewelry about the person became distinctly tarnished by a sulphurous emanation from the skin: a suggestive fact in connection with the question of ptomaines.

Dr. G. E. DE SCHWEINITZ.—I have had the opportunity of studying a number of cases of ordinary migraine, and some examples of typical ophthalmic migraine. The most remarkable series of hemicranias which have come under my observation, and which should probably be classed as ophthalmic, are those recorded by Dr. Weir Mitchell and myself, in which the prodromal visual disturbance took the form of an apparition. As these cases have already been published in detail, I shall not now refer to them further.

In a certain number of my cases of migraine, hemianopsia, numbness of the mouth, and numbness of the left upper extremity preceded the attack. In one such case I made examinations of the eyegrounds during the paroxysm, without, however, observing phenomena worthy of special record. In two of these cases ascending doses of cannabis indica, pushed to the point of tolerance, afforded the greatest relief. Another curious symptom that I have noted, in addition to the depression which follows intense pain, is that during and after the attack a mental state, amounting to melancholia, appears. In one such case after the paroxysm the patient is in a condition in which he fears that he either has done or will do some great wrong. I do not know whether this is the result of depression from great pain, or whether it is part of the nerve-explosion. As Dr. Wood has said, you may build as many theories in regard to the cause of migraine as you choose, but they fall like houses of cards. One theory, which has been much dwelt upon in modern times, is that all forms of migraine depend upon imperfect ocular balance and disturbances of vision. That these frequently are the origin of a host of violent headaches no one can deny, but that they alone are the cause of the complex phenomena which make up an attack of ophthalmic migraine, or indeed of ordinary migraine, is at least doubtful. It should be remembered that patients the subjects of migraines sometimes have two headaches: one, the hemicrania, as in a case recently seen with Dr. Weir Mitchell, in which the paroxysms were preceded by numbness and scintillating scotomata, and in which general treatment is efficient; and a second, usually persistent, frontal or occipital headache, the result of eye-strain or insufficient ocular muscles, and which is cured by the appropriate ocular therapeutics. I do not believe that the various forms of heterophoria, or refraction-error, cause true ophthalmic migraine, nor have I ever seen a case of this kind cured by their correction alone, however much this may have aided in the favorable result. I have investigated a number of

cases of migraine and epilepsy, and have never found any association, nor have they seemed to have had a common cause.

HYSTERICAL AMBLYOPIA IN A COLORED GIRL NINE YEARS OF AGE.

Dr. G. E. DE SCHWEINITZ.—I wish to report a case of amblyopia of hysterical origin occurring in a colored girl nine years of age. The child was brought to the dispensary of the Hospital of the University of Pennsylvania because she claimed that the left eye was sightless. The right eye was slightly myopic, otherwise healthy, and with the correcting lens the vision rose to normal. In the left eye there was a perfectly normal oval optic disc, healthy in color, and the retina natural. The refraction was hypermetropic. The child denied even light-perception, although the pupil reacted normally to the changes of light and shade. Remembering the observation of Bernutz, that the conjunctiva in hysterical cases is frequently anæsthetic, I tested this in the child under discussion, and found it as insensitive as if the eye had been cocaineized. A further examination revealed complete left hemianæsthesia. The ordinary tests with prisms, as well as that one proposed by Dr. Harlan, of this city, readily demonstrated the presence of vision in the eye in which light-perception was denied. The child had had no disease except measles; the mother, a mulatto, was healthy. The treatment consisted in the administration of some water colored with the compound tincture of cardamom, care being taken to impress upon the parent as well as the child the importance of giving exactly the dose, the impression being conveyed that the medicine was most powerful in its action.

The case gradually improved, and now, after a number of months, vision almost normal in amount has returned to the eye for which previously blindness had been claimed. It was practically impossible to make any determination of the color-fields of the right eye, or of the left eye, since the patient has admitted a return of vision.

It is not always easy to classify correctly that form of blindness which is called hysterical, because, to quote the able paper of Dr. Hill-Griffith, "it is sometimes doubtful, as some one has remarked, if the subjects should be considered as patients or as culprits." I should like to hear the opinion of the members of the Society on this point, and on any other points which may help to explain this curious form of amblyopia.

I have made the following interesting experiment in these cases, namely, in one case of simulated blindness (malingering) the subject was made to understand, by having the tests explained to her, that those present at the examination fully understood that her statements in regard to a lack of sight in the eye under examination were totally false. She was sufficiently intelligent to appreciate that she had been detected in her attempt at deceit, and readily admitted the charge. On the other hand, a perfectly intelligent woman, the subject of hysterical amblyopia, in whom the tests demonstrated the fact that the eye claimed for blindness had full visual acuity, utterly denied the possibility of sight, in spite of the fact of her apparent perfect appreciation of the sufficiency of the tests which had demonstrated that she could see.

Dr. CHARLES K. MILLS reported a case of

EMBOLISM OF THE RADIAL ARTERY.

The following case was seen in consultation with Dr. J. J. Healy: The patient, fifty-two years old, had symptoms of hepatic and pulmonary congestion. In about three weeks, when he had nearly recovered, but while still in bed, he was taken suddenly with intense pain in the front of the arm between the median line and the radial border, about two inches below the elbow. The pain extended to the forearm, hand, and fingers, which became practically helpless. All the fingers, with the exception of the little finger, were blue and cold, as were also, but to a less extent, the entire hand and forearm upward to the site of the initial pain. The greatest pain, blueness, and coldness were in the thumb and middle finger; they were like "dead fingers," and were almost completely paralyzed. All movements of the fingers, hand, and wrist, but particularly those of flexion, were much impaired, and slight swelling was present, but no real œdema.

In about twenty-four hours the lividity had considerably abated, and in forty-eight hours circulation was fairly re-established. The patient, however, continued to suffer greatly and was in a highly nervous state.

He was first seen by me ten days after the onset. A small swelling could be felt at the position of the first pain; the radial pulse on this side had disappeared, and the closed, cord-like vessel could be easily traced. He had great pain on pressure, following the line of the vessel, and considerable but less hyperæsthesia over the entire radial half

of the forearm; as the ulnar border was approached, the pain and hyperæsthesia diminished and disappeared. He complained greatly of feelings of coldness and pain here, and also in the thumb and fingers, except the little finger. Gradually the circulation was more fully restored, and the pain and weakness subsided. At the time of my last visit to the patient, four weeks after the occurrence of the embolism, he still complained of some coldness, tingling and pain in the hand, especially in the thumb, which continued to be more paretic than any other part. The treatment employed was chiefly local warmth, counter-irritation, internally anodynes and tonics. For the present stage, massage has been recommended.

The points which seem most worthy of consideration by a neurologist are the cause and characteristics of the pain, the nature of the paralysis, and the proper treatment. Embolism of the cerebral arteries apparently causes but little pain, doubtless because of the absence of nerves in the cerebral substance. In the extremities, and largely elsewhere, nerves, sensory or mixed, are in close relation with the arteries; the radial nerve, for instance, closely apposed, lies to the outer side of the artery. The persisting pain and hyperæsthesia in these cases are much as in a true neuritis.

DISCUSSION.

Dr. WHARTON SINKLER.—Some years ago I had the opportunity of seeing a lady who had several attacks of embolism of vessels, the result of extreme disease of the mitral valve. The first attack in which I saw her occurred while she had her arm elevated combing her hair. The right arm suddenly dropped motionless; there was violent pain in the arm; the limb was cold, and there was no pulse at the wrist or at the bend of the elbow. The power of motion returned on the following day; the radial pulse did not return.

Some months later she had embolism of one of the cerebral vessels, giving rise to temporary right hemiplegia and aphasia. Later there was another attack, but I do not recall the vessel involved. The first attack occurred some six years ago, and the patient died only a few days ago; but I do not know what was the cause of death, as she died in another city.

Dr. WHARTON SINKLER reported "A Case of Pericarditis occurring during an Attack of Acute Chorea in a Child Nine Years of Age."

Asylum Notes.

THE TREATMENT OF THE ACUTELY INSANE IN GENERAL HOSPITALS.

Last year a revised code of lunacy laws, under the name of the Gallup Lunacy Bill, passed the New York Legislature, and only failed to become a law by the opposition of the Governor. The same bill is to be introduced again at the present session. The New York Neurological Society, at its January meeting, appointed a committee, consisting of Dr. Frederick Peterson, Dr. Charles L. Dana, and Dr. Ralph L. Parsons, to examine and report upon this proposed new law. After commenting favorably upon some very excellent features of the bill, and after vigorously opposing the method of commitment prescribed therein, this committee closed its report at the February meeting with a number of recommendations, among which one in particular is deserving of careful attention, because of the novelty of the suggestion. We refer to the following: "A clause should be introduced into the bill providing that nothing in the lunacy laws of the State shall be construed to interfere with the reception and treatment of acute cases of insanity in chartered general hospitals, in the same manner and under the same conditions as patients suffering from other diseases are there received and treated, provided such hospitals have suitable accommodations approved by the State Commission in Lunacy."

In our opinion, this is the most valuable portion of the committee's report, for it suggests a step forward in the line of a great reform. The day of huge aggregations of persons with chronic and acute insanity in the palatial caravansaries known as asylums, where the mere attendance to the physical wants of the patients is often deemed sufficient therapeutics, is about to pass away. The insane are no longer to be considered in the light of dangerous criminals, and asylums are not always to bear the stigma of existing as a species of jail. What is the fate of a person with acute, curable insanity—one that could recover in from three weeks to three months—when sent to one of these "cathedral" institutions? His personality is entirely lost in the horde of from six hundred to two thousand mad people among whom he is placed. The superintendent, usually busy with the farming and plumbing, seldom has time to see the patients. A young assistant physician, commonly of small experience, takes the patient in hand along with the two hundred that he is to see twice daily. He cannot spend more than three hours if he will with the two hundred patients, because the clerical work required of him consumes

not only most of his day, but part of his night. The patient is considered, not as an individual, sick and requiring treatment, but in relation to the other patients of the ward. Does he disturb others? Then narcotize him. If that is impossible, put him into the pandemonium known as the "back ward." There his sick brain, before haunted only by his own phantasmagoria, beholds materialized the hideous specters of his imagination. And it is doubtful if any one in delirium has ever seen aught to compare with the waking nightmare of a "back ward" in some asylums.

Doubtless most asylum authorities do all in their power to improve the environment of their charges as far as is possible under present conditions, but proper individualization must necessarily be unattainable in such a concourse of people and with such small assistance. Hence it is that of late the question of radical reform in the present methods of caring for the insane has become more and more prominent. They are hereafter to be treated, at least in the earliest stages of their aberration, like other sick persons, only with greater delicacy and care, because the most complex and sensitive organ of their bodies is the one that is diseased.

We read of the provision of reception-houses in New South Wales and Queensland, and of lunacy wards in public hospitals in Victoria, for the treatment of insanity in its early stages. A psychopathic hospital with a hundred beds is about to be built in London, the administration of which is not to differ from that of a general hospital. The staff is to consist of a resident medical officer of asylum experience, and assistant, four visiting physicians, a consulting surgeon, an ophthalmologist, an aurist, a laryngologist, a gynæcologist, and a pathologist. A still later step in the direction of reform is the organization of an out-patient department at the West Riding Asylum, near Wakefield, England, which is calculated to change the present routine line of action completely with regard to the early treatment of the insane poor.

With these facts in mind, we cannot speak too favorably of the action of the committee of the New York Neurological Society. Their proposition to place it in the power of the sixty-three chartered general hospitals of this State to open special wards for the reception of the acutely insane, under the same conditions precisely as other classes of patients are received, would lead to vast improvement in the early and efficient treatment of the nutritive disorders of the brain. It would create a number of reception wards

in various parts of the city and State, where there is now absolutely no place for such purpose. Bloomingdale is overcrowded and about to be removed from the city. The method will lead to greater individualization, a deeper scientific study of insanity, and the training of nurses and practitioners for the better recognition and care of insane patients in their own homes, and many will recover without having attached to their name and reputation the inevitable stigma of having been in an asylum.—*N. Y. Med. Journal*, Feb. 22, 1890.

Book Reviews.

A TREATISE ON HEADACHE AND NEURALGIA, including Spinal Irritation and a Disquisition on Normal and Morbid Sleep. By J. Leonard Corning, M.A., M.D., Consultant in Nervous Diseases to St. Francis' Hospital, the Hackensack Hospital, etc., etc. With an Appendix: EYE-STRAIN, a Cause of Headache. By David Webster, M.D., Professor of Ophthalmology in the New York Polyclinic; Surgeon to the Manhattan Eye and Ear Hospital, etc. E. B. Treat & Co., Second Edition.

No physician of average intelligence can fail to extract enjoyment from the perusal of this, the second edition of Dr. Corning's book on Headache and Neuralgia. For several years past this accomplished physician has devoted much attention to the practical management of pain; indeed, no one among the present generation of physicians in this country, it is safe to say, has written or accomplished as much in this important field. We say accomplished, since the author of this volume is not only endowed with keen perceptive power and rare originality, but likewise with a forcefulness and lucidity of style which facilitates the transfer of knowledge, and renders the perusal of his writings rather a pleasure than a task.

Space does not permit us to review in detail this excellent volume; but we have no hesitation in saying, that in no other monograph on headache with which we are acquainted is so much that is original and practical to be found. Many of the suggestions regarding the management of pain, whether intra-cranial, extra-cranial, spinal or neural in its origin, is in the highest degree original and suggestive.

As regards Dr. Webster's appendix on Eye-Strain as a cause of headache, we have likewise only words of commendation to offer.

The first chapter of this part of the book treats of "Headaches dependent upon Errors of Refraction;" the second deals with "Headaches dependent upon Impaired Accommodation;" the third is devoted to "Headaches dependent upon Insufficiency of the Extrinsic Ocular Muscles;" and the fourth and final chapter gives a series of cases illustrative of the efficiency of "Graduated Tenotomy of the Ocular Muscles," when the conditions are such as to warrant operative interference of this sort.

In contradistinction to much of the recent literature on this subject, Dr. Webster's statements are eminently cautious and conservative. While cheerfully recognizing the importance of eliminating morbid conditions of the eyes as an adjunct in the treatment of minor functional disturbances, he is careful not to jeopardize the authority of his position by that short-sighted exaggeration which is the true birthmark of a weak judgment.

While cordially recommending this excellent treatise, we heartily congratulate both Dr. Webster and Dr. Corning on the admirable manner in which each has performed that portion of the allotted task for which he is so admirably suited.

PRACTICAL ELECTRICITY IN MEDICINE AND SURGERY.

By G. A. Liebig, Jr., Ph.D., and George H. Rohé, M.D.
Illustrated. F. A. Davis, Publisher. 383 pages. Price, \$2.00 net.

This most excellent book is divided into three parts. Part I. is devoted to physics and, as well, discusses the various forms of electrical apparatus likely to be of use to the general physician.

Part II. is physiological. The variations of reaction in disease and the diagnostic value of these modifications is here discussed.

Part III. is therapeutical.

The authors make no claims to originality, and state that they "have endeavored to place in the hands of the student and practitioner an intelligible account of the science of electricity and a trustworthy guide to its applications in the practice of medicine and surgery." The work undoubtedly deserves a place on the bookshelves of every doctor who desires to inform himself on electricity. To be read carefully as to its physics, physiological and diagnostic data. The instruments recommended are certainly the best in their way. The authors at least deserve credit in not pushing theoretical deductions as to therapeutical value to absurd length. This part of the book is concise, and contains the latest accepted views as to the practical application to various morbid conditions. So much rubbish has been allowed to enter this part of books on electricity that the reviewer had hoped that positive and original clinical observation by the author would have made it more concise in some parts and more at length in others. The book is certainly, however, a scientific guide and will not mislead the student or disappoint the physician. It is well printed and fully illustrated.

THE INTERNATIONAL MEDICAL ANNUAL AND PRACTITIONER'S INDEX FOR 1890. Edited by P. W. Williams, Secretary of Staff, assisted by a Corps of thirty-six Collaborators, European and American. Six hundred octavo pages. Illustrated. Cloth, \$2.75: E. B. Treat, Publisher, 5 Cooper Union, New York.

This is the eighth yearly issue of the handy reference one-volume annual. It perpetuates the well-earned reputation of the preceding issues. We especially commend the special departments of thermo-therapeutics by Percy Wilde, M.D. The careful resumé in the field of electro-therapeutics by A. D. Rockwell, M.D.; Sanitary Science by D. S. Davies, M. B. Lond. D. P. H. Cantab, and the various special departments of neurological medicine. It is truly of great practical value to the medical practitioner, and deals with new ideas and is abreast of the times, saving much time in journal reading. There is a slight increase in its size, but its cost remains the same.

PRACTICAL LESSONS IN NURSING. 12mo, cloth. \$1.00 each. Published by J. B. Lippincott Co., Philadelphia.

They comprise so far several very interesting little books for professional men to read, especially for the busy specialist to obtain valuable and practical knowledge in other fields of medical practice than their own. The latest is on "Diseases and Injuries of the Ear, their Prevention and Cure." By Charles Henry Burnett, A.M., M.D. There is certainly no one more capable to write upon this subject than he. The work is in no need of criticism, and praise is unnecessary. We commend it to our readers who desire to be informed on the subjects treated in a concise, direct and readable form.

THE LITERARY DIGEST, a Weekly Summary of the Current Literature of the World. Funk & Wagnalls, Publishers, 18 Astor Place, New York. Subscription price, \$3.00, ten cents a copy.

The first number is commendable and interesting. In this busy age, and especially to the physician, a periodical of this kind is a boon. The best thoughts, investigations and discussions appear in current periodical literature; and to have this epitomized by able men is a desideratum. We wish the journal all success.

BOOKS RECEIVED.

PRACTICAL PHOTO-MICROGRAPHY BY THE LATEST METHODS. By Andrew Pringle, F.R.M.S. New York, 1890: The Scovill & Adams Company, Publishers.

THE NEUROSES OF THE GENITO-URINARY SYSTEM IN THE MALE, with Sterility and Impotence. By Dr. R. Ultzmann, Professor of Genito-Urinary Diseases in the University of Vienna. Translated by Gardner W. Allen, M.D., Surgeon in Genito-Urinary Department, Boston Dispensary. Philadelphia and London, 1890: F. A. Davis, Publisher. \$1.00 net.

TRANSACTIONS OF THE AMERICAN NEUROLOGICAL ASSOCIATION, 14th Annual Session, held at Washington, D. C., September 18, 19, and 20, 1888. Published by "Journal of Nervous and Mental Disease." Price, \$1.00.

ST. BARTHOLOMEW'S HOSPITAL REPORTS, Vol. XXXV., 1889. Edited by W. S. Church, M.D., and W. J. Walsham, F.R.C.S. London: Smith, Elder & Co., Publishers.

PAMPHLETS, PERIODICALS, ETC., RECEIVED.

SEVENTH ANNUAL REPORT OF PHILADELPHIA POLYCLINIC AND COLLEGE FOR GRADUATES IN MEDICINE, embracing Polyclinic Hospital, the College Department, and the Second Annual Report of the Ladies' Aid Society of the Polyclinic Hospital (1890).

MINERAL SPRINGS OF THE UNITED STATES. By Judson Daland, M.D.

INTERMEDIATE TRACHELORRHAPHY. By H. J. Boldt, M.D. Reprint from Vol. XIV., Gynecological Transactions, 1889.

A CASE OF ALEXIA (Dysanagnosia). By Dr. Swan M. Burnett. Reprint from "Archive of Ophthalmology," Vol. XIX.

THIRTIETH ANNUAL REPORT OF STATE ASYLUM FOR INSANE CRIMINALS, Auburn, N. Y.

TWENTY-SECOND ANNUAL REPORT OF THE NEW YORK ORTHOPEDIC DISPENSARY AND HOSPITAL, 1889.

THE INSANITY OF DOUBT. By Philip Coombes Knapp, A.M., M.D. Reprint "American Journal of Psychology."

- METHODS OF EXAMINATIONS IN MEDICO-LEGAL CASES INVOLVING SUITS FOR DAMAGES. By Philip Coombes Knapp, A.M., M.D. Reprint from "Boston Med. and Surg. Jour."
- THE NIGHTINGALE. Edited by Sarah E. Post, M.D. New York.
- CONTRIBUTIONS TO ORTHOPÆDIC SURGERY, FOURTH FASCICULUS. By John Ridlon, A.M., M.D.
- ENUCLEATION OF TUBERCULOUS GLANDS. By Thos. W. Kay, M.D. Reprint from the "Medical Register."
- THE SEVENTEENTH ANNUAL REPORT OF THE METROPOLITAN THROAT HOSPITAL FOR THE TREATMENT OF DISEASES OF THE NOSE AND THROAT. New York.
- THE HOME-MAKER. Edited by Marion Harland and Grace Peckham, M.D.

Miscellany.

[Reprinted from the Proceedings of the American Association for the Advancement of Science, 1889, p. 26.]

REPORT OF THE COMMITTEE ON ANATOMICAL NOMENCLATURE WITH SPECIAL REFERENCE TO THE BRAIN.

During the past year, some of the members of the Committee have given the subject intrusted to them as much time as their regular duties would permit. They agree upon one point, viz., the advantages, other things being equal, of *mononyms*, (single word terms) over *polyonyms* (terms consisting of two or more words). Before making specific recommendations or presenting a final report, the Committee think it advisable that they and other anatomists should have an opportunity of discussing at leisure the simplified nomenclature which they are informed is employed in certain treatises which will be published during the coming winter. They therefore ask to be continued.

BURT G. WILDER, *Chairman*.
 HARRISON ALLEN,
 FRANK BAKER,
 HENRY F. OSBORNE,
 T. B. STOWELL,

Committee.

Note by the Chairman.—The treatises referred to in the above Report are Leidy's "Human Anatomy," and the following articles in Wood's: "Reference Handbook of the Medical Sciences," vol. viii., by E. C. Spitzka, "Spinal Cord" and "Histology of the Brain;" W. Browning, "Vessels of the Brain;" S. H. Gage and B. G. Wilder, "Anatomical Terminology;" B. G. Wilder, "Anatomy of the Brain," "Malformations of the Brain," and "Methods of Dissection," etc.

PRELIMINARY REPORT OF THE COMMITTEE ON ANATOMICAL NOMENCLATURE, ADOPTED DEC. 28, 1889, BY THE ASSOCIATION OF AMERICAN ANATOMISTS WITHOUT DISSENT.

"The Committee recommend:

1. That the adjectives DORSAL and VENTRAL be employed in place of *posterior* and *anterior* as commonly used in human anatomy, and in place of *upper* and *lower* as sometimes used in comparative anatomy.

2. That the cornua of the spinal cord, and the spinal nerve-roots, be designated as DORSAL and VENTRAL rather than as *posterior* and *anterior*.

3. That the costiferous vertebræ be called THORACIC rather than *dorsal*.

4. That the *hippocampus minor* be called CALCAR; the *hippocampus major*, HIPPOCAMPUS; the *pons Varolii*, PONS; the *insula Reilii*, INSULA; *pia mater* and *dura mater*, respectively PIA and DURA."

Signed by all the members.

JOSEPH LEIDY, *Chairman*.
HARRISON ALLEN,
FRANK BAKER,
THOMAS B. STOWELL,
BURT G. WILDER.

Thomas Dwight was added to the Committee.

The Committee desire frank and full expressions of opinion from scientific and medical journals, from individuals who receive copies, and from any others who are interested in the subject.

BURT G. WILDER, *Sec'y*.

NOTICE.

TENTH INTERNATIONAL MEDICAL CONGRESS.

To be held in Berlin, August 4th to 9th.

The Committee of Organization of the Tenth International Medical Congress, R. Virchow, *President*; E. von Bergmann, E. Leyden, W. Waldeyer, *Vice-Presidents*; O. Lassar, *Secretary-General*, have appointed the undersigned members of an American Committee for the purpose of enlisting the sympathy and co-operation of the American profession.

We are assured that the medical men of our country will receive a hearty welcome in Berlin. The Congress promises to prove of inestimable value in its educational results, and in securing the ties of international professional brotherhood. It is most important that the American profession should participate both in its labors and its fruits.

Delegates of American Medical Societies and Institutions, and individual members of the profession, will be admitted on equal terms. The undersigned, therefore, beg to express their hope that a large number of the distinguished men of our country will appreciate both the honor conferred by this cordial invitation and the opportunity afforded us to fitly represent American medicine.

The Congress will be held at Berlin, from the fourth to the ninth of August.

The arrangements in regard to a few general meetings and the main scientific work, which is delegated to the sections, are the same as in former sessions. A medico-scientific exhibition, the programme of which has been published a few weeks ago, is to form an ingredient part. It is to the latter that the Berlin Committee is very anxious that both the scientific and the secular press should be requested to give the greatest possible publicity.

The office of the Secretary-General is Karlstrasse, 19, N. W., Berlin, Germany.

S. C. BUSEY Washington, D. C.	WM. T. LUSK, New York,
WM. H. DRAPER, New York.	WM. OSLER, Boston, Mass.
R. H. FITZ, Boston, Mass.	WM. PEPPER, Philadelphia, Pa.
H. HUN, Albany, N. Y.	J. PEYRE PORCHER, Charleston, S. C.
A. JACOBI, New York.	J. STEWART, Montreal, Can.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

HYPNOTISM ;
WITH A CRITICISM ON SOME RECENT EXPERIMENTS AT
LA SALPÊTRIÈRE.

By FREDERIC BATEMAN, M.D., F.R.C.P.,

Senior Physician to the Norfolk and Norwich Hospital; Corresponding Member of the Academy of Medicine of Paris; Hon. Member of the New York Neurological Society; Corresponding Member of the Psychiatrial Society of St. Petersburg, etc.

PERHAPS no branch of physiology has received so much attention of late years as the localization of cerebral faculties; and thanks to the researches of recent observers, among the most distinguished of whom are some of our own countrymen, the localization of function stands out in bold relief from the ordinary course of events, as marking an epoch in the history of medicine, to which there is nothing parallel in modern times; but although the motor region of the cortex cerebri has been mapped out with marvellous precision and exactitude, a less satisfactory result has been attained in reference to the localization of sensorial centres, and this remark applies especially to the visual centre; for, although, thanks to the labors of Ferrier, Schäfer, Sanger-Brown, and others in this country, and of a host of scientific workers on the Continent and in America, a flood of light has recently been thrown upon it, I am justified in saying that in the year of grace 1890, the most conflicting views prevail in reference to this branch of cerebral physiology—the exact localization of the visual centre.

I can readily imagine that some of my readers may say, What can the visual centre have to do with hypnotism and with the Salpêtrière experiments? I trust to be able to show that the subject I am considering has a direct bearing on the physiology of vision, and the communication which I venture to make to this journal may be deemed as supplementary to an essay on the visual centre which I have lately published in a scientific journal, "*Les Archives de Neurologie*," edited by M. Charcot.

Perhaps few subjects have attracted more attention lately than hypnotism, or the production of artificial sleep, and as some remarkable experiments made at La Salpêtrière have recently been brought under my notice, I have been induced to make them the subject of a communication to this journal, and I am especially induced so to do, as I do not agree with the inference that the originators of these experiments have sought to deduce from them; and here I would remark that by the influence of Charcot and Richet at Paris, Bernheim and Beaunis at Nancy, and Hack Tuke in this country, men of science are turning their attention to the study of hypnotic phenomena, instead of leaving them to charlatans and unscientific observers.

As the result of Professor Charcot's experiments in hypnotism, two conditions have been described, called respectively "hysterical lethargy" and the "cataleptic state."

It is reported that most cases of grave hysteria can be thrown into the first of these two conditions by directing the eyes to be fixed steadily on some point—the tip of a penholder held in the hand, for instance; in a few moments the head inclines to the right or to the left, the eyelids close, the limbs become motionless and limp, but the power of speaking remains. This is the condition of hysterical lethargy in which, amongst other symptoms, there is marked neuro-muscular excitability, a phenomenon first observed by M. Charcot, and which he designated "*hyper-excitabilité neuro-musculaire des hypnotiques*," and which consists of the aptitude of voluntary muscles to contract under the influence of simple mechanical irritation.

It is easy to cause the patient to pass from the hypnotic condition to the cataleptic stage. All that is required is to open the eyelids and allow the retinae to be stimulated by the rays of light; the patient immediately becomes cataleptic, but a remarkable difference is observed between the two sides, as shown by the following experiment made by M. Lepine, one of M. Charcot's assistants.

A female patient is hypnotized; if asked to speak, write or make any gesture whilst in this condition, she obeys. The left eyelid is then opened and the left retina stimulated, thus plunging the right hemisphere into a state of catalepsy; there is no change from a linguistic point of view; the patient continues to speak, to write and to gesticulate. The process is modified; the left eyelid is closed and the right opened with corresponding stimulation of the right retina, thus plunging the left hemisphere into a state of catalepsy; immediately all communication with the external world is suppressed, the patient can neither speak, write, nor gesticulate. "Le masque facial reste muet." A moment before, when the right hemisphere was in a cataleptic condition, every form of language subsisted; now that a similar condition is induced in the left hemisphere, language of every kind is abolished.

The above experiment was repeated in the case of another patient who knew by heart some pieces of poetry. Whilst in the hypnotic state, she repeated certain verses distinctly. The left eyelid was raised, but she continued her recitation; but on the left lid being closed and the right opened, thus producing catalepsy in the left hemisphere, she straightway ceased to speak in the middle of a verse, sometimes even in the middle of a word. On closing the right eyelid, she began her recitation again at the very place at which she left off, recommencing sometimes in the middle of a word.

M. Ballet, in commenting upon the above curious phenomena, lays great stress upon their value as experiments *in vivo*, tending to confirm the results of clinical observation, which would place the seat of speech in the left anterior lobe to the exclusion of the right. Now, I am not

intending to enter upon the question as to whether the speech-centre, if there be one, is situated in this or that part of the brain; I have discussed this subject at considerable length elsewhere. This is not the time or the place to discuss the general question of the localization of speech, but I simply moot the question as to whether the deductions drawn from these experiments are not open to doubt. I think they are; at all events they are certainly not beyond the reach of fair and legitimate criticism.

In the first place, although the results claimed for them are so startling and of such momentous importance, I am not aware that they have even been confirmed by subsequent observers. I find that my scepticism is shared by no less an authority than Professor Bernheim of Nancy, who, in a private communication to me, attributes the phenomena observed to "suggestion." "It has been thought," says he, "at La Salpêtrière, that the hypnotized, whilst in a state of lethargy, are unconscious and do not hear; whereas they hear everything, and often strive to guess what the observer is desirous of eliciting from them. A hypnotized person who has never witnessed Charcot's experiments, and who is not aware of what is desired of her, will certainly never exhibit the phenomena described. It is certainly an affair of *suggestion*; the experiments are misleading, and can in nowise help to solve the difficult questions connected with the pathology of aphasia."

My friend, Dr Hack Tuke, whose researches upon this subject are so well known, has favored me with a letter in reply to my inquiry, in which he says that he has never been able to convince himself that hypnotic experiments, which appear to support the alleged function of Broca's convolution, are free from that subtle and constant source of fallacy—unintentional suggestion. In an article on "Artificial Insanity," in the "Journal of Mental Science" for 1865, and also in his "Influence of the Mind upon the Body, with especial Reference to the Imagination" (1872), Dr. Tuke has insisted on the numerous influences of imagination and suggestion in so-called animal magnetism and hypnotism.

Furthermore, whilst engaged in writing this paper, my attention has been called to a report of a curious illustration of the effects of hypnotic suggestion, lately witnessed at St. James' Hall, London. It is stated that a mesmerist threw a gentleman, named King, into a mesmeric trance so profound that the passage of a needle through the fleshy part of his arm caused no sensation. In this state, Mr. King gave humorous lectures and very curious imitations of Mr. Gladstone, Mr. Irving, Mr. Grossmith, and Dr. Parker. He was told by the mesmerist that he was each and all of these persons, and, quite unconsciously, he accepted the responsibility: the will became the slave of a suggestion; an automaton was substituted for the true volitional self.

Moreover, Professor Charcot himself, as I have elsewhere stated, admits that hysterical mutism can be produced artificially by hypnotism; and in the appendix to the third volume of his "*Diseases of the Nervous System*," one of his pupils, M. Cartaz, thus describes in detail how, by hypnotic suggestion, hysterical mutism is produced: During the period of somnambulism the patient is made to converse; the observer, then lowering his voice, says to her: "I don't understand you. What do you say? Why, you can't speak," and in an instant the patient is aphasic, being unable to speak or to phonate. This, says M. Cartaz, is the exact representation of the mental disturbance observed in hysterical patients.

Whilst, therefore, recognizing the scientific interest attaching to these curious experiments, I cannot admit that the inference that has been drawn from them is a logical one; and I think they should not be cited as confirmatory of the localization of speech in the left hemisphere.

I am not at all sure that there may not be also an anatomical objection to the deductions which have been drawn from these experiments.

In a former paper I have entered at some length into the consideration of the different theories of the visual centre, and I have shown that the whole subject is far from being definitely settled, as the most divergent notions exist as to

the course of the fibres at the optic chiasma, between it and the mesocephalic ganglia, and again between these and the cerebral cortex.

Moreover, Charcot's assumption, that there is a supplementary crossing of the fibres of the optic tracts in the corpora quadrigemina, by which all fibres from one retina would pass to the opposite hemisphere, is by no means recognized as absolutely correct; indeed, Charcot himself speaks of it as only an hypothesis, not based at present on any anatomical grounds, but nevertheless supplying a ready means of presenting, in a very simple form, the rather complex facts revealed by clinical observation.

I need scarcely point out that if there be no other decussation of the optic fibres but that which is said to take place at the chiasma, stimulation of one retina should affect both hemispheres equally, and the Salpêtrière experiments would lose all their import—that is, assuming that the prevailing opinion is the correct one—that the crossing of the fibres at this point is only partial. I am aware that this partial decussation is not admitted by all anatomists, and that the course of the optic fibres is still a matter of dispute: in fact, certain German anatomists, Biesiadecki, Mandelstamm, and Michel maintain that the fibres of the optic nerves undergo complete decussation at the chiasma, as occurs in fishes, amphibia, reptiles, and birds; and that the semi-decussation is to be looked upon simply as a theory, which, however, explains the facts observed in clinical medicine. I find that Ferrier also, in describing Charcot's scheme, characterizes it as unsatisfactory, and in contradiction with now well-established clinical as well as experimental facts.

It is therefore clear that the exact course of the fibres of the optic nerves is still undefined, and perhaps no question of late years has excited so much controversial discussion as the exact determination of the visual centre. The whole subject of the course of the fibres of the optic tract, and of the nervous connections of the retina with the brain, is discussed in an exhaustive manner by Professor Grasset, of Montpellier, who rejects the schemes both of Charcot and of his pupil Féré, for which he substitutes one of his

own, frankly admitting, however, that it is purely hypothetical, but may be useful as a means of graphically representing the actual state of our knowledge of this difficult subject of cerebral semeiology. M. Grasset recognizes three decussations of the optic fibres :

1st. Semi-decussation at the chiasma, where the internal fibres cross, whilst the external fibres continue in a direct course.

2d. The external fibres cross in the neighborhood of the corpora quadrigemina, so that by this arrangement the decussation is complete, and thus all the optic fibres from one eye are reunited in the internal capsule of the opposite hemisphere.

3d. The external fibres undergo a further decussation, beyond the internal capsule, before terminating in the convolutions of the hemisphere, and by this means each occipital lobe will contain the external fibres from the eye of the same side and the internal fibres from the opposite eye.

M. Grasset adds that this triple seat of partial decussation seems to be indispensable for the proper interpretation of certain clinical facts ; as to the exact locality in which it occurs, he suggests that it may be in the corpus callosum. He admits that the above arrangement is somewhat complex, but that the facts to be explained are of a no less complex character also.

Another author, Michael Foster, in writing upon this subject, says that the nervous centre is not a double centre with two completely independent halves, one for each eye ; there is a certain amount of communion between the two sides, so that, when one retina is stimulated, both pupils contract. The authors of the Salpêtrière experiments, of course, assume that one retina only is stimulated, and only one hemisphere plunged into catalepsy.

Besides the conflicting opinions as to the points of decussation of the optic nerves, there would seem to be a further objection to the acceptance of the inference drawn from the Salpêtrière experiments in reference to the localization of the faculty of language. In order to establish any connection between these experiments and the localization

of speech, it is necessary to assume that the fibres of the optic tract find their way into the anterior lobes of the brain, the supposed seat of speech—which is by no means universally admitted.

The course of the fibres of the optic tract between the external geniculate body and the cortex of the brain is not well ascertained, and is still the subject of scientific inquiry; at all events, a great diversity of opinion exists in reference to it. Gratiolet asserted that the optic tract is directly connected with every part of the cerebral hemisphere in man, from the frontal to the occipital region; and Professor Hamilton, of Aberdeen, in a communication to the Royal Society, on "The Cortical Connection of the Optic Nerves," expressed the same opinion.

In treating of this subject, that careful and accurate observer, Dr. Ross, says that both anatomical and physiological, as well as pathological observations, make it certain that most, if not all, of the fibres of the optic tracts terminate in the cortex of the occipital lobes, but that the course of the fibres in the intervening space between the cortex and the external geniculate body, is the subject which has excited the greatest controversy.

From a private communication with which Dr. Gowers has favored me, he speaks most emphatically upon this point, and says that his views of the physiology of the cortex would exclude altogether the idea of any passage of fibres from the optic tract to the motor speech region.

I have thought it desirable thus to enter at great length into the consideration of the bearing the experiments made at La Salpêtrière, upon persons in a state of hypnotism, may have upon the localization of functions, but I must think that our knowledge of the exact construction of the visual centre, of the precise distribution of the fibres of the optic tract, and of the relation of the two visual centres to each other, is at present so imperfect as not to justify the originators of the above interesting experiments to quote them as evidence of the localization of speech in any part of the left hemisphere of the brain.

A STUDY OF CEREBRAL PALSIES OF EARLY LIFE, BASED UPON AN ANALYSIS OF ONE HUNDRED AND FORTY CASES.*

By B. SACHS, M.D.,

Professor of Nervous and Mental Diseases at the New York Polyclinic,

AND

By F. PETERSON, M.D.,

Lecturer in the Department of Nervous and Mental Diseases at the New York Polyclinic.

FEW diseases are better known or more thoroughly understood than infantile spinal paralysis. Its clinical symptoms and its pathology have been definitely determined, so that poliomyelitis anterior acuta scarcely needs further study. The very opposite is true of infantile *cerebral* palsy. While there is but a single form of disease included under the term infantile spinal paralysis, we are forced to admit that there are several different forms of cerebral palsy. The attempt to fix upon certain cases of cerebral paralysis in children and to label them infantile cerebral palsy as a direct analogy of the better known spinal disease has led to great confusion, although we shall see that there is some justice in drawing this analogy. Nor is it entirely correct to speak of cerebral *spastic* palsies as distinguished from *atrophic* paralysis, for we shall report at least two cases in which the element of spasticity was entirely wanting, although the cases were undoubtedly of cerebral origin, the proof of which was furnished by the post-mortem examination in one of these two cases.

The large majority of the cases with which we are here concerned represent spastic forms of paralysis and as regards the distribution of the palsy may very properly be

* Read at the stated meeting of the New York Academy of Medicine, April 3, 1890.

divided into cases of spastic hemiplegia, of double spastic hemiplegia or diplegia, and of spastic paraplegia. Since the clinical subdivisions are so easily made, it would seem to be a curious fact that these cases have been so poorly understood and so little studied. There were many reasons for this: first and foremost, the great difficulties in obtaining autopsies, the majority of these cases either living on to a very advanced age, or else dying in almshouses, where no interest was taken in them; then again, the condition was so frequently associated with idiocy; or the individuals were regarded as hopeless cripples that did not possess sufficient interest to repay careful study. And lastly, the term infantile cerebral palsy proved to be a stumbling-block. A number of cases and some few autopsies were reported, disclosing a variety of lesions; there seemed to be little hope of bringing order out of chaos. The truth of the matter, that we had a number of different forms of disease and a variety of pathological processes to consider, was a long time forthcoming. We hope to show by this paper that much of the confusion that has surrounded this subject will be removed if we consider that a variety of morbid symptoms may give rise to any of the three forms of paralysis, and that the character of the paralysis will depend upon the site and extent of the morbid lesion. But for the difference in the areas of the brain affected and the degree of irritation or destruction of brain substance, the symptoms in all these cases would be very much the same.

The subject which we present has a live interest at this present time, and yet it is nearly fifty years ago since the first work in this field was done. In 1842, Prof. Henoeh wrote his inaugural dissertation, "*De Atrophia Cerebri*," and gave an excellent account of infantile cerebral paralysis.¹ Heine² referred to these cases in a monograph on spinal paralysis of children, published in 1860. Little³ was well acquainted with them. In 1868, Benedikt,⁴ the neurologist, described them. The French schools soon took up the subject, and Cotard,⁵ Wuiilaumier,⁶ Bourneville,⁷ wrote important papers on spasmodic infantile paralysis, some of

them referring to the anatomical features of these diseases. As recently as 1883, several English authors (among them Hadden⁸ and Ross⁹) published the clinical details of a small number of cases and the accounts of a few autopsies.

Two publications have stirred up the recent discussion on this question. The first was Kundrat's¹⁰ monograph on porencephalus, in which this one morbid state was carefully studied. The second was Strümpell's¹¹ paper in 1884, in which he suggested that infantile cerebral hemiplegia was due to a polioencephalitis acuta. This single statement which was decidedly original, but had no post-mortem proof, has raised a great hue and cry which have not yet subsided. Whatever other good it may have accomplished, Strümpell's theory has at least imbued an old subject with new life. His article was quickly followed by a number of valuable contributions, among which those of Ranke,¹² Bernhardt,¹³ Wallenberg,¹⁴ Kast,¹⁵ Jendrassik and Marie,¹⁶ Gowers¹⁷ and Hoven¹⁸ are by far the most important of the European contributions. In America, able articles have been written by Dr. Sarah McNutt,¹⁹ Drs. Sinkler,²⁰ J. Lewis Smith,²¹ Knapp,²² Lovett²³ and Gibney.²⁴ Chief and foremost of all is an exhaustive monograph by Prof. Osler,²⁵ in which 151 new cases were analyzed with a skill which characterizes all of Prof. Osler's work.

There would seem to be some need of an apology after this for the study of another series of cases. Our first reason for doing this is, that the work preparatory to this paper was begun several years ago, before the publication of Prof. Osler's monograph. Secondly, we had some views of our own to advance; and, lastly, the unusual number of cases at our disposal called for special elaboration.

The cases here reported upon were, with very few exceptions, examined by one of the authors of this paper. We are greatly indebted to Dr. Gibney and Dr. Townsend for referring the greater number of these cases to us from the Hospital for the Ruptured and Crippled. Dr. Gray was kind enough to allow us to use the notes on cases seen in his department. Ten of the cases were seen in private practice.

The physician is no longer content, or at least should not be, to make the diagnosis of apoplexy; of hemiplegia, or of paraplegia, in the adult. It is his aim to determine whether the special form of paralysis be due to hemorrhage, thrombosis, embolism, tumor, abscess, or what not. In short, he studies the symptoms of each case with special reference to pathology of the disease. And so with infantile palsies: it is not enough to recognize spastic hemiplegia, diplegia or paraplegia, but the attempt should be made to determine the special morbid condition underlying each form. The large number of cases made it imperative upon us to make a distinct effort in this direction.

In view of the small number of autopsies recorded in literature this effort may seem hazardous; but a single autopsy in a well-observed case is a guide in the study of dozens of others; and with the advances made in a knowledge of cerebral lesions, the neurologist is fortunate in being able to reason with a great degree of certainty from clinical symptoms to the morbid lesion which they indicate.

A thorough knowledge of the clinical symptoms is, however, the starting point of this study. In order not to weary you with the details of these cases, we give you the conclusions to be drawn from our entire series with reference to each point at issue. And we shall give but a few histories in extenso that you may recognize the chief types of which we treat. Before proceeding to these cases, we submit a table showing that of the 140 cases* there were 87 males and 53 females; there were 105 hemiplegias, 24 diplegias, and 11 paraplegias.

TABLE I.—Showing the sex and form of paralysis in 140 cases of infantile cerebral palsy.

<i>Form of Paralysis.</i>	<i>Males.</i>	<i>Females.</i>	<i>Total.</i>
Right Hemiplegia	37	15	52
Left Hemiplegia	26	27	53
Diplegia	15	9	24
Paraplegia	9	2	11
Total	87	53	140

* Since the above was written we have seen at least fifteen additional cases, but we have concluded not to alter the tables in the body of this paper.

HEMIPLEGIAS.

CASE I.—(No. 48.) A. F., æt. four and a half years, male, first child, difficult labor and instrumental delivery. From very first day right hemiplegia. Slight athetosis and associated movements. Contracture at elbow, formerly pes equino-varus, improved by operation. All reflexes of right side lively. Mental condition fair.

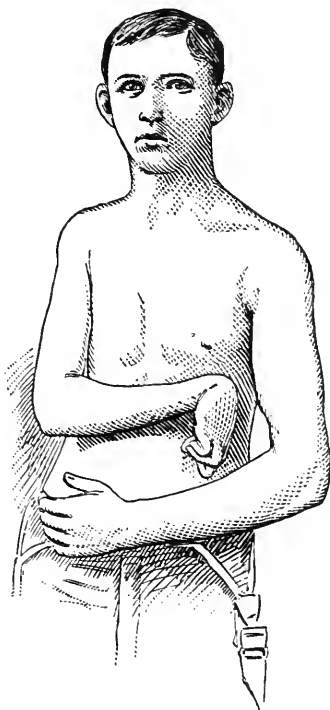


Fig. I.—CASE IV., No. 138. Right hemiplegia with contracture and retarded growth of arm.

CASE II.—(No. 58.) L. B., female, æt. nine years. Congenital left hemiplegia. Labor normal; fourteenth child; movements of child ceased a few days before birth; expected to be still-born; weighed four pounds at birth. From age of two years epileptic attacks every two to four weeks. Fine associated movements of both sides. Arm, leg, and face of left side involved. Very great retardation of growth of arm and leg; some contractures of flexors of arm and wrist; imbecile; microcephalic.

CASE III.—(No. 44.) L. E., female, æt. eight years; acquired hemiplegia; onset at age of five years; convulsions for nine hours and coma; could not move, talk or walk for three months. Face and leg have recovered considerably; very great retardation of growth of arm. Marked associated movements. Contracture of right arm; reflexes of right side exaggerated.

CASE IV.—(No. 138.) J. K., male, æt. seventeen years. Right hemiplegia at eight years of age following typhomalarial fever; was delirious and unconscious during nineteen days; no convulsions. After recovering from coma, right arm, face, and leg were found paralyzed. Complete aphasia and entire loss of memory of everything occurring before typhoid. Had to be re-educated. Athetoid and associated movements. Reflexes exaggerated right side. Enormous contracture of flexors of right hand and fingers and great retardation of growth of right upper extremity. Right leg somewhat smaller than left; right talipes valgus; asymmetry of face. Electrical reactions and sensation entirely normal. Has recovered speech fully and is bright, but several years behind others in education. *Fig. I.*

DIPLEGIAS.

CASE V.—(No. 141.)* J. O., female, æt. sixteen. Congenital diplegia; mother kicked in abdomen by horse two months before birth of child and made unconscious thereby. Three other children, all healthy. Tedious labor, no instruments used, no fits or convulsions. Did not attempt to creep or walk; teeth at usual age. Patient has menstruated since tenth year and was weak in back, arms and leg from earliest childhood. Extreme spastic contracture of adductors and flexors of thighs; double talipes varus, equinus on right side. Left arm worse than right. Athetoid movements of left hand. Has frog walk. Intelligence good. *Fig. II.*

CASE VI.—(No. 31.) M. L., male, æt. three years. Congenital diplegia. Asphyxiated during labor. Mother had pneumonia, and died five days post partum. Rigidity of arms, legs and back. Hands did not unclinch for two years. Frequent convulsive seizures alternately of right and left

* This case, although under observation for a long time, was omitted from our list by mere accident. The history is sufficiently characteristic to deserve special mention.

side, including face. Cannot talk, walk or stand. Feeble minded; cross-legged position and all reflexes exaggerated.



Fig. II.—CASE V., No. 141. Diplegia; double talipes equino-varus, athetosis of left hand.

PARAPLEGIA.

CASE VII.—(No. 50.)* C. F., male, æt. one year. Congenital paraplegia. First child, labor hard and dry for forty-eight hours. Asphyxiated. From first day up to age of six and a half months child had a rapid succession of tonic and clonic spasms affecting all the muscles of the body, causing rigidity of all extremities, opisthotones with extreme arching of back, enormous exaggeration of all reflexes, ankle and quadriceps clonus on slightest excita-

* While this article was passing through the press, this child died. An autopsy was obtained by Dr. L. E. Holt. A careful study of the pathological findings will be made and published in due time. But one other autopsy on infantile paraplegia has hitherto been published.

tion. Convergent strabismus; crying continually, Mental condition probably imbecile. Epileptic spasms controlled slightly by bromide treatment. *Fig. III.*



Fig. III.—CASE VII., No. 50. Paraplegia. Photographed in epileptiform convulsion.

CHIEFLY MONOPLÉGIA.

CASE VIII.—(No. 47.) H. K., male, æt. twenty-two months. Acquired right hemiplegia; onset at six months following convulsions during pertussis and pneumonia. Trace of weakness in right arm; distinct spastic paralysis of right leg; knee-jerk exaggerated; feeble-minded. This case made at first the impression of a monoplegia.

FLACCID PARALYSIS.

CASE IX.—(No. 61.) M., female, æt. two years at time of death. First-born of healthy parents. During the fifth month of pregnancy mother was thrown from a carriage without sustaining any serious injuries. The child born at full term apparently normal in all respects. At age of two to three months a general listlessness and nystagmus were observed. During its entire life child was unable voluntarily to move any muscle of its body. All muscles extremely flaccid, but all reacted perfectly to electrical currents. There was not the first symptom of any mental awakening. During the first year of its life child noticed light, but later on absolute blindness set in. There was a developmental defect of the optic nerves, which was reported upon by Dr. Knapp, a similar condition of the nerves having been observed in only two other cases. Hearing seemed to be acute. There was unusual hyperexcitability of auditory and tactile impressions. The child never had convulsions, not even during dentition; no rigidities. All reflexes lively.

Speech was, of course, entirely wanting. The child died of pneumonia following bronchitis. The autopsy will be referred to later on. This case was made the subject of a special paper by one of us²⁶ (S.), and entitled "On Arrested Cerebral Development, with Special Reference to Cortical Pathology." This paper was a study of some of the cortical changes giving rise to idiocy, but we have since come to learn that the report of the case and the autopsy have a wider significance than was attributed to them at the time.

CASE X.—(No. 80.) H. M., male, æt. eleven and three-quarter years. The second of three children; an uncle said to have been similarly affected. Asphyxiated at birth; instrumental delivery. Began to teethe late; teeth have rotted away. Made imperfect attempts to walk at fourteen months; crept around on his buttocks; both feet turned inward, right more than left; learned to talk, but mind has always been very feeble. Has had frequent epileptic attacks, grand and petit mal. Both upper extremities excessively weak, but no rigidities. Lower extremities poorly developed. All muscles respond to faradic current, but some of them so feebly that very strong currents are needed. Knee-jerks weak but present; boy's father is a teacher who has done much with his defective mind.

From the records herewith presented to you, you will infer that much as the cases differ from one another, they also have much in common; they yield a distinct composite portrait. The child is either born with, or in its early life develops some form of paralysis; a hemiplegia, a diplegia, or a paraplegia. In the congenital cases there has been some disturbance during pregnancy, or labor has been tedious and difficult or definite cause cannot be given. In the acquired cases we have seen that the onset of paralysis may occur after acute infectious diseases, during convulsions, or from causes that cannot be fathomed. In the majority of cases there is marked spasticity and extreme contractures; in two cases there is a flaccid form of paralysis; in the last case reported, the knee-jerks and other reflexes were weak; in all other cases the reflexes were exaggerated, at least on the side or sides paralyzed. Some show peculiar associated athetoid or other post-hemiplegic movements; in all, there was more or less retardation of growth, and all stages of mental impairment were found from weak-mindedness to

complete idiocy ; a few, however, are of good mental development. No changes in sensibility were observed, and the electrical reactions were never markedly altered.

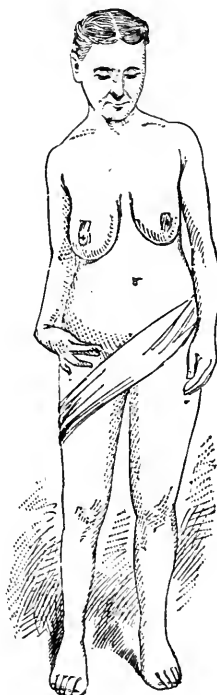


Fig. IV.—Right hemiplegia, from age of 9 months, in a woman 36 years. Contracture and retarded development of paralyzed side.

These cases and some of the symptoms they exhibit are referred to by the poet, historian, and the painter. The Duchess of Gloucester, according to Sir Thomas Moore, had much ado in her travail. Her son, "Richard III., came into this world feet foremost. Shakespeare makes "Richard" say of himself:

"Deformed, unfinished, sent before my time
Into this breathing world, scarce half made up,
And that so *lamely* and unfashionable
That dogs bark at me as I *halt* by them."

In Raphael's Transfiguration, the demoniac boy has characteristic athetoid position of one hand.

It is now in order to see whether our cases shed any new light on the understanding of these palsies, and what relation the facts hold to the commonly received classification into three distinct subdivisions.

TABLE II.—Showing the age at onset.

<i>Age at Onset.</i>	<i>Hemiplegia.</i>	<i>Diplegia.</i>	<i>Paraplegia.</i>	<i>Total.</i>
Congenital.....	22	20	7	49
During 1st year.....	27	1	2	30
“ 2d “.....	17	1	—	18
“ 3d “.....	16	2	1	19
“ 4th “.....	4	—	—	4
“ 5th “.....	4	—	—	4
“ 6th “.....	2	—	—	2
“ 7th “.....	1	—	—	1
“ 8th “.....	5	—	—	5
“ 9th “.....	—	—	—	—
“ 10th “.....	—	—	—	—
Under 15 years.....	4	—	—	4
Unascertained.....	3	—	1	4
Total.....	105	24	11	140

From the above table we learn that of 105 cases of hemiplegia 22 are congenital; of 24 cases of diplegia 20 are congenital, and so are 7 or possibly 8 of the 11 cases of paraplegia. Diplegias and paraplegias are more likely to be of congenital origin, hemiplegias are more apt to be acquired in the first three or four years after birth; but it is well worth noting that there are 22 congenital cases of hemiplegia (over 20 per cent. of all cases of hemiplegia); some of the cases noted as occurring in the first year may be congenital, and this would help to swell the percentage of congenital hemiplegias. It will not do, therefore, to make the broad distinction so frequently made on the basis of the acquired or congenital character of these palsies. Our table is in happy agreement with the one given by Osler as regards hemiplegias (15 of 120 were congenital). Sixty of our hemiplegic cases were developed before the close of the third year of life; then there is a distinct falling off up to the age of ten; between ten and fifteen we have a few more; in three cases the age could not be ascertained. Will you also note that some of the acquired

cases of paraplegia or diplegia have occurred after the age of three years, and one or the other of these cases may have been congenital.

TABLE III. —Showing age at examination.

<i>Age.</i>	<i>Hemiplegia.</i>	<i>Diplegia.</i>	<i>Paraplegia.</i>	<i>Total.</i>
Under 4 years.....	40	16	8	64
Between 4 and 10 years.....	26	6	2	34
“ 10 and 20 “.....	14	2	1	17
“ 20 and 30 “.....	18	—	—	18
Under 40 years.....	7	—	—	7
Total.....	105	24	11	140

This table records the ages at examination, from which it is apparent that diplegia and paraplegia are comparatively short-lived while hemiplegias often attain a very considerable age. Statistics of the exact ages at death would be more accurate; but inasmuch as our material includes cases from every kind of institution, even from pauper asylums, the inferences to be made are tolerably correct.

TABLE IV. —Showing causes given in 91 cases of acquired cerebral palsy.

<i>Causes Given.</i>	<i>Hemiplegia.</i>	<i>Diplegia.</i>	<i>Paraplegia.</i>	<i>Total.</i>
Convulsions.....	20	1	—	21
Pneumonia.....	6	—	—	6
Trauma to Head.....	6	—	—	6
Pertussis.....	4	—	—	4
Measles.....	2	2	—	4
Scarlatina.....	3	—	—	3
Onset with fever.....	2	—	1	3
Hereditary Syphilis.....	2	—	—	2
Cerebro-spinal Meningitis.....	2	1	—	3
Onset with fever and convulsions only.....	2	—	—	2
Fright.....	2	—	—	2
Hydrocephalus.....	—	—	2	2
Vaccinia.....	1	—	—	1
Typho-malarial Fever.....	1	—	—	1
Small-pox.....	1	—	—	1
Tonsillitis.....	1	—	—	1
Epileptic Seizure.....	1	—	—	1
Gastro-enteritis.....	1	—	—	1
Unascertained.....	26	—	1	27
Total... ..	83	4	4	91

TABLE V.—Showing ascertainable causes in 49 cases of congenital cerebral palsy.

<i>Causes.</i>	<i>Hemiplegia.</i>	<i>Diplegia.</i>	<i>Paraplegia.</i>	<i>Total.</i>
Instrumental delivery, tedious labor...	4	1	1	6
Ante-partum trauma to mother.....	2	3	—	5
Premature birth.....	1	2	1	4
Asphyxia at birth.....	—	3	—	3
Asphyxia in twin birth.....	—	1	1	2
Tedious labor, breech presentation...	2	—	—	2
Primipara, dry birth tedious labor...	—	—	1	1
7 mos. child, dry birth (48 hours)...	—	1	—	1
Primipara (æt. 45), tedious labor...	—	1	—	1
Maternal fright (ante-partum).....	1	—	—	1
Uræmia of mother.....	1	—	—	1
Pneumonia of mother (died 5 days post-partum), child asphyxiated.....	—	1	—	1
Convulsions of mother during pregnancy, difficult labor	—	1	—	1
Mother in fever for 10 weeks, ante-partum.....	1	—	—	1
Unascertained.....	10	6	3	19
Total...	22	20	7	49

In these tables we have analyzed, as far as possible, the causes in 91 acquired cases and 49 congenital cases of cerebral palsies.

Among the acquired hemiplegias the acute infectious diseases, including pertussis and pneumonia, play a very important role; a strikingly large number have come on during convulsions; in these cases the convulsions are not the initial convulsions of acute infectious diseases; the latter are considered separately in Table VI. The cases that have come on with fever and convulsions are noted in addition; but not wishing statistics to prove more than

TABLE VI.—Showing the relation of convulsions to the onset of the palsy.

Cases in which convulsions apparently preceded or were associated with the palsies occurring in—

Pertussis.....	3
Pneumonia.....	2
Scarlatina.....	1
Dentition.....	1
Vaccinia.....	1
Baptism.....	1
Fright.....	1
With beginning of menstruation.....	1
Fall on head.....	1
Gastro-enteritis.....	1
Total.....	13

Apparently immediate symptom of a focal lesion—in 2 cases.

Idiopathic and apparently only cause of palsy—in 20 cases.

Palsy occurring in ordinary epileptic seizure—in 1 case.

they should, it is but fair to add that in many of these cases fever may have been present, and that from among these 20 cases we may allow that several, if not all, show the onset Strümpell claims for his cases of polioencephalitis. We ask you also to note that in 6 cases of hemiplegia there was a distinct history of traumatism, that 2 hemiplegias and 1 diplegia were the result of cerebro-spinal meningitis; 2 cases of diplegia came on after measles, a fact of some importance. In 26 cases of hemiplegia we were not satisfied with the statements elicited, and have therefore marked them as "causes unascertained." The table of causes in the congenital cases points a moral. In 16 cases of the 49, say in 33.3 per cent., there was some difficulty in labor, simple delay or instrumental delivery. The older writers, Little, Gaudard,²⁷ and others refer to this cause, but have tolerably favorable statistics, Little mentioning but 4 cases; Wallenberg gives 6 of 160 cases, and Osler 9 of 97 cases. The authors mentioned referred to hemiplegia only, and speak of forceps delivery as the element of danger. Our percentage is higher, because we include all forms of cerebral paralysis, and tedious labor as well as instrumental delivery. The moral is, that the forceps should be applied, if necessary, or delivery hastened by other means if protracted labor can be averted. A child's brain and skull have a wonderful power of resistance, but do not credit them with greater virtue in this respect than they really possess. The mother's life is by far the more important, but it is well to reflect that other things being equal she prefers a child that is neither paralyzed or idiotic.

As regards the mode of onset, of the congenital cases, it would appear paradoxical to say anything; but in several cases which were distinctly congenital, attention was first drawn to the disease by the appearance of convulsions at an early day. In these cases the convulsions are due to the same lesion or process which is responsible for the palsy. In the acquired cases convulsions preceded the onset of the other symptoms in 36 of 83 cases of hemiplegia, and in one case of acquired diplegia; loss of consciousness generally accompanies the convulsions; in 6 cases there was a dis-

tinct onset without loss of consciousness or convulsions; this occurred in 4 cases of left hemiplegia and in 2 of right hemiplegia; 2 of these left hemiplegias were distinctly syphilitic.

TABLE VII.—Analysis of six cases in which there was onset without loss of consciousness or convulsions.

<i>Hemiplegia.</i>	<i>Age.</i>	<i>Cause.</i>
Right with aphasia.....	2 years.....	Tonsillitis.
Right.....	10 mos.....	Unknown.
Left.....	2½ years.....	Syphilis.
Left.....	8 “.....	Unknown.
Left.....	1½ “.....	Syphilis.
Left.....	2¼ “.....	Fall on head.

Strümpell has made the onset with convulsions and fever a distinctive feature of his cases. The 6 cases referred to in our list answer to his description of infantile cerebral palsy as regards the hemiplegic form of paralysis. It is more natural to infer that they are not cases of polio-encephalitis than that they are anomalous cases of that class.

On this subject of initial convulsions and loss of consciousness a word should be added, even at the risk of anticipating some inferences regarding the pathology of these palsies. Initial convulsions and loss of consciousness are distinctly cortical symptoms. They indicate very considerable cortical disturbance either by direct injury or by severe or sudden injury to any part of the brain, which would also imply disturbance of the cortex as of every other part of the brain. Relatively small injuries to the cortex, hemorrhages or cortical encephalitis, will bring about loss of consciousness and convulsions; relatively large injuries to the interior need not exhibit these symptoms. The exception is in cases of embolism or sudden hemorrhages in which the suddenness of the shock disturbs the entire brain. This argument was urged by one of us²⁸ (S.) in a paper published some years ago in which a typical case of capsular hemorrhage was diagnosticated mainly on the line of argument just referred to; and in examining statistics of Wallenberg and others with reference to this point, we find that convulsions occurred only in cases of

embolism anywhere, and in all cortical affections, however slight these may have proved to be. If all cases of infantile hemiplegia were cases of polioencephalitis corticalis, convulsions would invariably be present, but such is not the case.

The *form* of paralysis has been frequently referred to. Hemiplegia, double hemiplegia or diplegia, and paraplegia speak for themselves. Table I. gives the relative number of cases of each form in our list. Monoplegias are not included, although other writers had reported some such cases. We have seen but one case in which we were tempted to make a diagnosis of monoplegia, and this case gave distinct evidence on closer examination that the arm as well as the leg had been involved in the earlier course of the disease. The march of the disease resembles adult hemiplegia in this that the leg recovers very much more quickly than the arm for reasons that need not be given here. Under the heading of diplegia we have classed all cases in which both upper and lower halves of the body were involved; in some of these one leg or one arm had so far recovered that the cases might have been interpreted as hemiplegia with an additional involvement of the other arm or leg; but here again on closer scrutiny we became convinced that at one time all four extremities had been affected.

The involvement of the face is a matter of some doubt. The majority of the cases were seen at a time long after the recovery of the face. We can vouch, however, for the following statements. The face was affected in 11 cases of right hemiplegia, in 9 of left hemiplegia, and in 2 cases of diplegia. In 2 cases (1 of right and 1 of left hemiplegia) the leg was worse than the arm; in all other hemiplegics the arm was the part more affected. In all diplegics the legs were more affected than the arms. Trunk and neck muscles were distinctly involved in two cases of diplegia. Strabismus occurred in one of right hemiplegia, in four of left hemiplegia, and in three cases of diplegia.

It has been doubted whether aphasia followed the same laws in the infantile hemiplegia as in the adult form, and

Bernhardt¹³ has seen fit to write at length on this special subject. Aphasia can only be said to be present if the hemiplegia comes on in an individual who had already acquired articulate speech, hence the 49 cases of hemiplegia and the 21 of diplegia which occurred before the age of two years are excluded from this consideration and of the remaining 56 cases of hemiplegia the fewest dispensary patients or their parents could give satisfactory statements. We have records of 17 cases of hemiplegia with undoubted aphasia. Of these 17, 10 were cases of right hemiplegia and 7 cases of left hemiplegia. Eight of these 17 cases were observed by one of us (S.) in private practice. Of these 8, 5 had been distinctly aphasic, and three of the 5 were cases of left hemiplegia. This relatively large proportion of aphasia in cases of left hemiplegia is rather striking by contrast with the adult cases. As we grow older we appear to become more and more left-brained. In earlier years both hemispheres are equally entrusted, so it seems, with this highest faculty of speech. Bernhardt also comes to the conclusion that aphasia in children accompanies right as well as left hemiplegia. Prof. Osler notes aphasia in 13 out of 120 cases of hemiplegia and only one of these with left hemiplegia. Wallenberg's statistics give 45 cases in 94 right hemiplegics and 17 in 66 left hemiplegics, but he includes all sorts of speech disturbances, and the statistics cannot well be utilized with regard to true aphasia. Defective speech was noted three times in our cases of right hemiplegia, twice in diplegia, and in most of our 80 cases of idiocy.

The reflexes are either lively or exaggerated in the large majority of cases. The exceptions are noted in Table VIII.

TABLE VIII. —Showing the condition of the deep reflexes (knee- and wrist-jerks) in 11 cases in which they were not exaggerated.

<i>Form.</i>	<i>Normal.</i>	<i>Diminished.</i>	<i>Absent.</i>	<i>Total.</i>
Hemiplegia	4	1	3	8
Diplegia.....	1	1	1	3
Total....	5	2	4	11

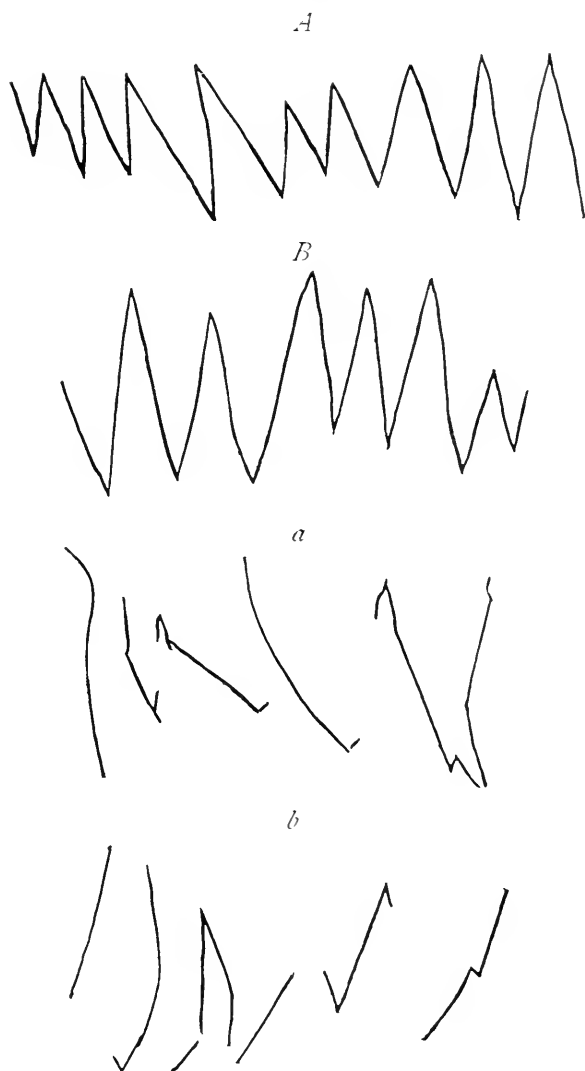
In all other cases the deep reflexes were exaggerated.

The points to be noted are these: The exaggeration of reflexes occurs in the parts paralyzed or paretic. The great excitability of the reflex is often the best proof to be had of the former involvement of the leg or arm, although bilateral exaggeration of the reflexes in cases of unilateral paralysis must be taken into account; owing to the extreme contractures of the opposing sets of muscles, the reflexes can frequently not be obtained. This is true particularly of the ankle clonus and triceps reflexes. We must note, however, that in several cases the knee-jerks and other reflexes were subnormal and in some cases absent. In one case (No. 50) the quadriceps clonus could be obtained.

Disturbances of motion, such as choreiform, athetoid associated movements are observed with unusual frequency after hemiplegia, and as we have learned also after diplegias in children.

In 105 cases of hemiplegia, choreiform movements occurred in	6
“ “ “ athetoid “ “	21
“ “ “ associated “ “	15
“ “ “ rhythmical “ “	1
“ “ “ ataxia “ “	1
“ “ “ tetanoid contractions “ “	1
In 24 cases of diplegia, choreiform movements occurred in	1
“ “ “ ataxia “ “	1
“ “ “ athetoid movements “ “	1
“ “ “ nystagmus “ “	2

These disturbances of motion after single and double hemiplegias have an unusual interest; they all point to some interference with the proper transmission of motor impulses either of the voluntary or of the inhibitory kind. The changes in the pyramidal tract are no doubt largely responsible for these peculiar movements, and that disturbances anywhere in the motor tract may bring about such aberrant movements is probable from the fact that these movements occur from the most diverse lesions, from lesions in the cortex as well as from lesions of the crus, as shown in an unpublished case of our own. Of associated movements we have seen a number of happy examples. In one instance the movements of the paretic arm and hand



A and B—Movements of the normal hand.

a and b—Movements of paretic hand.

following the movement of the opposite side were so considerable that we were able to take tracings exemplifying this fact. Westphal²⁸ has offered the theory that in health there is a tendency for both upper extremities to act con-

jointly, but that inhibitory impulses permit unilateral and single action. In disease this inhibition is removed, and therefore the effort to move one arm or hand results in the movement of both sides. The irradiation of impulses, therefore, enters primarily into this question, but it seems to us to be open to doubt whether this irradiation occurs in the brain or in the spinal centres. In every case we must call in cortical inhibition to explain the separation of these irradiating nerve currents during normal life. Greidenberg has gone very fully into this special topic. (*Arch. f. Psych.* vol. 17).

Rigidity with contracture is one of the cardinal features of all these palsies, and the deformity resulting therefrom, as a rule, leads the patients or their relatives to seek medical advice. With the exception of the flaccid cases before noted, a very considerable number go on to marked contracture of one or several groups of muscles. Table IX.

TABLE IX.--Showing the forms of contracture in the various cerebral palsies.

<i>Form of Contracture.</i>	<i>Hemiplegia.</i>	<i>Diplegia.</i>	<i>Paraplegia.</i>	<i>Total.</i>
Flexors of elbow.....	29	—	—	29
“ “ both elbows	—	3	—	3
“ “ carpus and fingers.....	23	1	—	24
“ “ knee.....	5	—	—	5
“ “ both knees	—	2	—	2
Extensors of knee.....	1	—	—	1
Adductors of thighs (cross-legged position).....	—	13	2	15
Talipes equino-varus	17	—	—	17
Double talipes equino-varus	—	5	2	7
Talipes equino-valgus.....	3	—	—	3
Talipes equinus	2	—	—	2

will give the main points, which need no further explanation except this, that attention should be drawn to the fact that cross-legged position of the legs occurs in diplegias and not exclusively in paraplegias, as is generally stated. Furthermore, that the contractures may be so extreme that the individual has a frog gait, as in the case of J. O., and that in three cases of hemiplegia there was distinct talipes equino-valgus, while in all other conditions there was either simple varus or equino-varus position.

Why flexor and not extensor muscles are the seat of contracture, adductors and not abductors, is a puzzling question. It is probably due to the mechanical principles involved in the construction of joints.

A word in passing with regard to trophic disturbances. In many of these cases the circulation in the skin of the paralyzed limbs is as poor as in infantile spinal paralysis. There is often the same livid hue of the palsied limb. In all cases occurring early in life, and particularly in those of congenital origin, there is apt to be a very marked retardation of growth. This was most distinct in nine cases of hemiplegia, three of diplegia, and three of paraplegia. Atrophy of the thenar eminence was observed in one case of diplegia, coming on after a cerebro-spinal meningitis.

The association of epilepsy with infantile cerebral palsies is perhaps the gravest feature of these diseases. In our experience it is the one danger to be feared, and should be considered most carefully in any case of cerebral palsy in a child. In our list, 62 out of 140 cases were afflicted with epilepsy, or 44.3 per cent. of all cases. There were among the hemiplegic cases 41 cases of general epilepsy, 9 of the Jacksonian type, and one case of petit mal (in all about 50 per cent.). In 24 cases of diplegia 7 had general epilepsy (29 per cent.), one had Jacksonian epilepsy. In 11 cases of paraplegia 4 had general epilepsy (about 36 per cent.). The percentage of epilepsy in our own list differs but slightly from that given by Gaudard, Wallenberg, and Osler. In view of the high percentages conceded on all sides, there can be but little doubt that taking all cases of ordinary epilepsy, a very fair proportion developed in connection with infantile palsies. Every sign of early palsy may have disappeared, while the epileptic taint remains. A case seen in private practice brings this out very forcibly :

E. B. (No. 81), girl seventeen years of age ; menstruated at age of eleven ; third of four children. All others died of acute infectious diseases. Father was fifty-six years of age at time of birth of child. Mother was thirty years younger. The child has had epileptic attacks every three to four months for some years. No convulsions during childhood. Had been treated for ordinary epilepsy by many physicians ; brominized for years. Closer inquiry revealed the fact that about four years ago the girl had an apoplectic attack, and on examination we found distinct evidence of left hemiplegia. Very marked weakness of left side ; greatly

increased reflexes ; slight mental enfeeblement and marked anæmia. Bromides were discontinued, the patient put on tonics, and is doing well, at least as well as under bromides, which had evidently deepened the mental apathy.

It will be seen that the percentage of epilepsy was greatest in hemiplegia, but that it was a distinctive feature of all forms of paralysis here considered.

Taking into account the cortical origin of a large number of these cases of epilepsy, it was natural to expect a higher percentage of Jacksonian epilepsies, but in most of these cases the original focal lesion has disappeared, and a general atrophy and sclerosis have been established. General and not localized epilepsy is the natural result of this change.

Together with the occurrence of epilepsy we should note the large number of cases exhibiting some form of mental enfeeblement. The conclusions on this point are presented in Tables X and XI, where we have attempted

TABLE X.—Showing the relation of mental defect to the age of onset also.

<i>Age of Onset of Paralysis.</i>	<i>Feeble-mindedness.</i>	<i>Imbecility.</i>	<i>Idiocy</i>	<i>Total.</i>
Congenital.....	6	15	14	35
Under 3 years	10	18	6	34
3-5 years.....	2	3	—	5
5-10 "	2	3	—	5
Over 10 years.....	1	—	—	1
Total.....	21	39	20	80

TABLE XI.—Showing the relation of mental defect to the form of palsy.

<i>Form of Mental Defect.</i>	<i>Hemiplegia.</i>	<i>Diplegia.</i>	<i>Paraplegia.</i>	<i>Total.</i>
Feeble-mindedness.....	16	2	3	21
Imbecility.....	31	7	1	39
Idiocy	7	8	5	20
Insanity (epileptic).....	1	—	—	1
Total.....	55	17	9	81

to distinguish between feeble-mindedness and imbecility and idiocy. Eighty of 140 cases exhibited some form of mental impairment ; 69 were either in congenital cases or in those acquired in the first three years of life ; 52 per cent. of the hemiplegic patients, 71 per cent. of the diplegic, and about 82 per cent. of the paraplegics were thus afflicted.

The worst form (idiocy) was most marked in the paraplegias (45 per cent.), and least marked in hemiplegia, being found in only 6.75 per cent. of all cases. Here again mental impairment can be said to be in proper relation to the extent of the cerebral lesion which is presumably less extensive in cases of hemiplegia than in the other forms of cerebral palsies; though we must allow that the later development of many of the hemiplegias may have something to do with these results.

As a point of special interest we wish to add that Little in 19 cases of paraplegias in which there was some impairment of mental condition found 13 feeble-minded or idiotic (68 per cent.) and 6 (32 per cent.) of good intelligence.

TABLE XII.—Analysis of *stigmata degenerationis* (exclusive of contractures) present in 57 cases of cerebral palsy in children.

	<i>Hemiplegia.</i>	<i>Diplegia.</i>	<i>Paraplegia.</i>	<i>Total.</i>
Microcephalus.....	21	4	2	27
Leptocephalus.....	19	1	—	20
Macrocephalus.....	4	—	1	5
Marked cranial asymmetry.....	25	—	2	27
Marked facial asymmetry.....	19	—	—	19
Cranium progonæum.....	5	2	—	7
"Gothic" palate.....	9	1	—	10
Imperfectly developed teeth.....	10	1	1	12
Supernumerary teeth.....	—	1	—	1
Hirsuteness.....	—	1	—	1
Neuropathic ear.....	1	1	—	2
Strabismus.....	5	3	—	8

As for cranial defects,²⁹ the table appended will give all the information we have, though it is well to note that in most cases of hemiplegia there is a flattening of the skull on the side of the lesion, and in almost all cases of cerebral palsy most of the cranial diameters are below the normal averages.

In this review of clinical symptoms we have shown that excepting the fact of paralysis, there are no symptoms peculiar to infantile hemiplegia that are not also found in diplegia and paraplegia. While the symptoms vary somewhat in degree in these different forms, all forms have all symptoms in common, excepting those of the onset. It is a difference of degree, not of kind. It remains for us to prove whether or not a study of pathological conditions

compels us to draw a distinction between these three forms, and whether or not hemiplegia, diplegia, and paraplegia respectively represent distinct morbid entities. We shall see that a variety of morbid lesions is to be found underlying these conditions, and that the same lesion or condition may in the one case be responsible for a hemiplegia, in the other for a diplegia, and so on. And furthermore, if definite forms of disease are to be diagnosticated, the diagnosis must rest upon other symptoms rather than the mere form of the paralysis. This branch of our inquiry is beset with great difficulties. In the scarcity of autopsies we have shared the fate of other writers. In spite of our very large clinical experience, we have but two autopsies of our own.* But we claim this one advantage that in both these cases the post-mortem findings were of very recent date—a great advantage, if we reflect that in most of the cases recorded in literature the conditions found were the final result of pathological processes which had continued for years, and which shed no light whatever upon the initial morbid lesion, and yet this is the salient point of the entire controversy. With the information gained by our macroscopical and microscopical studies we have analyzed the records of 105 autopsies including our own. This list could have been enlarged if we had had access to the publications of Richardiere³⁰ and some others. Our list, however, in-

TABLE XIII.—Analysis of pathological findings in hemiplegia, diplegia and paraplegia, based upon the most recent autopsies, including Wallenberg's and Osler's cases, but not those of Richardiere or Audry.

<i>Lesions.</i>	<i>Hemiplegia.</i>	<i>Diplegia.</i>	<i>Paraplegia.</i>
Atrophy, sclerosis and cysts.....	40	19	—
Porencephalus.....	2	4	—
Hemorrhage.....	23	—	—
Embolism.....	7	—	—
Thrombosis.....	5	—	—
Agensis.....	1	1	—
Tubercle.....	1	—	—
General cortical sclerosis.....	—	—	1
Total.....	79	24	1

Not including 64 cases of hemiplegia with porencephalus, and 32 cases of bilateral porencephalus collected by Audry.

*A third autopsy has been added since the above was written (cf. note p. 301).

cludes all the recent cases,* and the report of these will atone for the omission of those described by the older writers.

The first and the most conspicuous feature of this table is the prominence given to atrophy, sclerosis, and cysts. All these are terminal conditions and are almost useless for the determination of the initial lesion. Cysts are no doubt frequently due to hemorrhages, and if this could be statistically shown, the number of cases due to hemorrhage would have been materially increased. Zacher has noted one case in which cystic formations occurred together with an osteoma, but the patient died thirty-three years after onset of lesion. Wallenberg has recorded an interesting case of cyst in the left peduncle.

Porencephalus is a secondary condition, and although much has been written on this subject, we know little of its origin. In some cases it is probably the result of arrested development, due to interference with foetal circulation (Kundrat); in others it may have been the result of early encephalitis or even intra-uterine cerebral hemorrhage. A number of different lesions may bring about a condition of porencephalus; moreover, the term has proved to be exceedingly elastic, and what one author has termed atrophy, another has called porencephalus. Audry,³⁷ for instance, has collected 64 cases of this condition, some of which in the other lists are labeled atrophy or atrophy and sclerosis. We are forced, therefore, to rely upon cases in which the initial lesion has not disappeared; and here the first fact that is brought out with great distinctness is that hemorrhage, thrombosis, and embolism, the conditions which give rise to adult apoplexy, are also found to be a frequent cause of the cerebral palsies of early life. There is mention in the table of a case of tubercle reported by Seeligmüller³⁸ It is hardly fair to include such a case, for it was one of general tuberculosis in the course of which a hemiplegia appeared. While no other similar cases have been published, it is evidence of faulty logic that Seeligmüller should

* Two cases of Kast, one by Hoven, Wallenberg, Salgo,³¹ Langenbeck,³² Fürbringer,³⁴ Zacher,³⁵ Hirt,³⁶ Peterson [cf. page 323 of this article], and Sachs.²⁶

have inferred from this one case that latent tuberculosis is almost the sole cause of infantile hemiplegia. His case simply shows that hemiplegia may appear together with other symptoms, but there can generally be little doubt as to the nature of the process. It is for this reason that we have excluded from our list a case of our own in which the autopsy revealed a tubercle of the quadrigeminal region which had given rise to a hemiplegia in the course of the disease.

It may be surprising to find that polioencephalitis of Strümpell is not referred to. It is time that we should define our position with regard to this question. First of all, in order to distinguish this from two other lesions which have been termed polioencephalitis inferior (progressive bulbar paralysis) and polioencephalitis superior (nuclear ophthalmoplegia), let us speak of this as polioencephalitis corticalis. What proof have we that there is such a condition? Anatomical proof, none; we are willing to concede, however, that some of the many cases of atrophy and sclerosis may have been due to this polioencephalitis, but it is unfortunate for Strümpell's theory that all of the autopsies made soon after the onset of the disease have shown other conditions, and not a polioencephalitis. But let us be charitable or just, and say that even these autopsies were not made in cases sufficiently recent. We must add, however, that cases which correspond very closely to the cases which Strümpell considered typical of polioencephalitis corticalis showed hemorrhage, embolism, etc., of recent origin. Strümpell says, however, that not all cases of infantile hemiplegia need be due to this cause, and that all authors have misinterpreted his views. Is there no probability, then, that a few or any of the cases of infantile hemiplegia are cases of polioencephalitis corticalis? There is some circumstantial evidence showing that there is a brain-lesion which would seem to be analogous to spinal palsy of children (poliomyelitis anterior). Möbius³⁹ gives the history of two children of one family, aged one and one-half and three years respectively, who were stricken down with fever, loss of appetite, and somnolence. One developed

a typical poliomyelitis of the upper extremity; the other, spasmodic hemiplegia without aphasia. This is striking clinical evidence, though some might claim it to have been merely a coincidence. Another proof: Strümpell has but very recently reported two cases of adult apoplexy in which every one would have made the diagnosis—and indeed he made it—of embolic softening, but the post-mortem examination revealed a condition of encephalitis hæmorrhagica of the gray as well as of the white matter. Marie¹⁶ who is inclined to support Strümpell, expressed the opinion in 1885 that the encephalitis would attack the white as well as the gray matter, and thinks that this would not destroy the analogy with poliomyelitis, for in that condition the white fibres are sometimes involved. Jendrassik and Marie favor the perivascular (inflammatory) origin of the condition of lobar sclerosis, which they have carefully described. In view of all this, we venture the opinion that polioencephalitis corticalis may be the cause of some of the cases of infantile palsies; but, we add, not of the hemiplegia alone, for we have seen several cases, including one seen by the courtesy of Dr. Holt, in which all the symptoms were those of Strümpell's disease, but there was a diplegic and not a hemiplegic form of palsy. In these cases the cerebral character of the symptoms was so distinct that a confusion with poliomyelitis was out of the question.

We insist that, until further pathological proof shall be forthcoming, polioencephalitis corticalis shall be diagnosed last, not first. In a short paper, published some years ago in the *JOURNAL OF NERVOUS AND MENTAL DISEASE*, one of us insisted that there was strong reason to think, by analogy with adult apoplexy, that the lesion might be the same in the infantile form. A short abstract of that history will show the analogy:

M. M. (Case No. 75), boy, two and one-half years old; one and one-half years previously pneumonia; tonsillitis with fever up to 102°. Four days after this, typical right hemiplegia and aphasia, positively without coma or convulsions. The onset was as typical as in the ordinary mild hemorrhage into the internal capsule in the adult, and the

progress of the disease and the mode of recovery (which became complete) were quite like what we see in adult cases.

In 1887 the evidence on this point was not so strong as it is now, and this leads again to the subject of thrombosis, hemorrhage, and embolism. These vascular troubles were generally considered to be peculiar to advanced age, with the exception of embolism, which every one is ready to concede may occur at any age in which heart-disease occurs. Thrombosis was thought frequent enough, from the occurrence of syphilitic endarteritis in children. Hemorrhage in adults is attributed to the existence of miliary aneurisms. In the child, miliary aneurisms have been found, and Prof. Osler^{25,4} has described a large aneurism of the anterior cerebral artery occurring in a boy six years of age. If atheromatous degeneration of the arteries be less frequent than in adults, another condition is found to which Recklinghausen refers in his book on the "Pathology of Circulation and Nutrition," page 84. This is a fatty degeneration in the wall of the cerebral blood-vessels. Little notice has been taken of this, as indeed of all else that pertains to the vascular pathology of children.

Meningeal and cortical hemorrhages are shown to be more frequent in children, while all other cerebral hemorrhages are more frequent in adults. This will explain the more serious character of the symptoms in the young, and the more frequent occurrence of epilepsy and mental impairment in children. As the prognosis would be materially affected according to the cortical or cerebral character of the lesion, we have sought for the point of differential diagnosis between lesions so situated. To this end the occurrence of convulsions is specially to be considered. In the paper referred to it was stated: "Loss of consciousness is an extremely variable symptom; it seems to depend rather upon the quantity of blood effused than upon the area involved. Not so with convulsions. A convulsion, if it is anything, is a cortical affair, the result of cortical irritation." To which should be added that the irritation may be direct or indirect; the sudden shock imparted to the

cortex by the occurrence of embolism anywhere in the brain is apt to cause convulsions, and a lesion in any part of the gray matter of the central nervous system may excite convulsions; but hemorrhage over the cortex, even if slight, is apt to be accompanied by convulsions. Hemorrhage into the interior of the brain, unless very large, and the condition of thrombosis—slow occlusion of a cerebral vessel—are more apt to be marked by absence of convulsions and possibly also by preservation of consciousness. In other words, if you can exclude embolism and sudden and very large hemorrhage, and lesion of the lower gray centres, the absence of convulsions at the onset of an apoplectic attack is in favor of a lesion in the interior of the brain rather than in or upon the cortex. Of this I am more certain still, that whatever may be the symptoms accompanying the onset of an apoplectic attack, if the convulsions reappear after the initial symptoms, the lesion is cortical, or else an additional insult has occurred. A possible exception might be made in favor of thalamus lesion. These statements are corroborated by the results of recent autopsies, though only a small number of cases refer to all the facts needed. A case observed and examined post-mortem by one of us (P.) is in point :

A. W., male, aged fifteen, bright at school, expert swimmer, at age of eight or nine years was in the habit of diving a distance of twenty to thirty feet from a railroad-bridge. Shortly after this began to suffer from intense headaches, which gradually grew worse, until he was twelve years old, when mental changes began to be apparent. Gradually loss of memory, confusion of mind, and steady progress toward dementia. At the same time moroseness, melancholia, morbid fear, and coprolalia. One month after admission into the Poughkeepsie Asylum had a severe epileptic fit. Two days later right hemiplegia with constant right-sided hemi-epilepsy; became comatose. The clonic spasms of the right side continued at intervals of several days, but gradually became limited to the right leg alone. Four days later, death. Autopsy showed the outer dural surface to be normal; on the right side the subdural space presented a limited pachymeningitis hæmorrhagica interna, but merely a thin organized detachable stratum of long

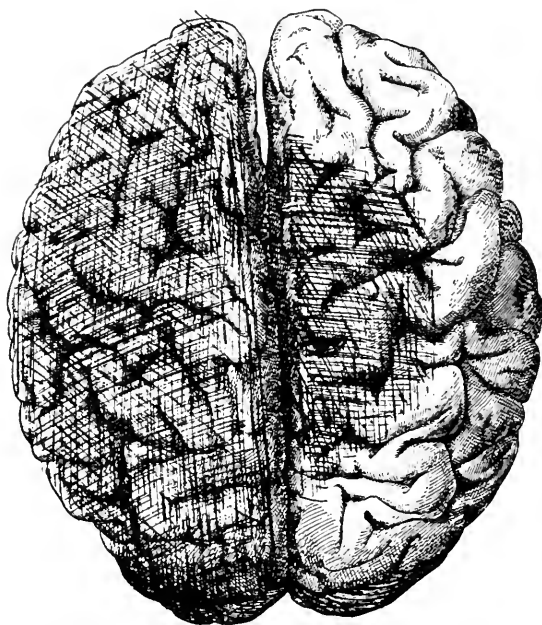


Fig. VI.—Showing extent of pachymeningitis hæmorrhagica over superior surfaces of hemispheres.

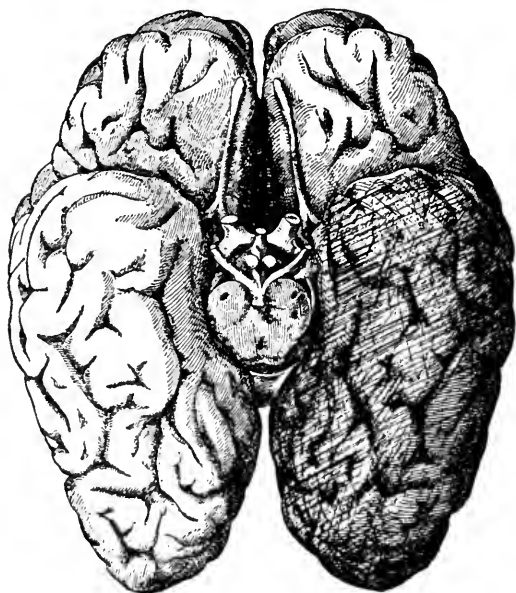


Fig. VII.—Showing extent of process on inferior surfaces of left hemisphere.

standing. Over the left side, however, the pachymeningitis was exceedingly widespread, extending over the whole surface of the hemisphere, both above and below, except in the anterior fossa of the skull. Over the convexity of the hemisphere the hæmatoma was very thick, consisting of strata of various ages, some of them undoubtedly dating from the beginning of his symptoms. Between two and three ounces of fresh coagula were spread on the convexity, especially in the Rolandic region. The extent of the pachymeningitis and hemorrhage is shown in the accompanying illustration. The left hemisphere was greatly compressed; the brain-substance itself seemed normal. The brain weighed $35\frac{1}{4}$ ounces. The ventricles were widened and distended with clear serum. There was no disease of or injury to the cranial bones. There was no lesion of any kind in substance of hemispheres, ganglia, pons, or medulla. *Figs. VI. and VII.*

Thrombosis occurs in a comparatively small number of cases. It is due either to the fatty change in the walls of the blood-vessels, or to syphilitic endarteritis, of which we have seen several cases, and Seibert⁴⁰ has described one, and is probably found in marantic cases. Gowers lays particular stress upon the thrombosis and occlusion of smaller cerebral veins, and thinks this the most important factor in the causation of infantile hemiplegia. All that we can say is that he must prove this to be true; the autopsies analyzed give no evidence whatever of this condition.

Thus far we have had reference chiefly to the pathological lesions of infantile hemiplegia. In diplegia nothing is said in our table of hemorrhage, thrombosis, and embolism, though it is more than likely that some of the cysts referred to were originally due to clots, and the occurrence of meningeal hemorrhages over both hemispheres giving rise to diplegias is well known. But these cases either die early before the form of paralysis is well established, or if they live for many years the initial hemorrhage has disappeared and the secondary conditions only remain. You will please observe, however, that what is properly termed agenesis corticalis occurs in a case of hemiplegia reported by Kast, and in the case of diplegia reported by one of us (S.) The

microscopical changes appear to have been very similar in both cases—in the one case unilateral, in the other bilateral, which accounts for the hemiplegia in the one case, for diplegia in the other.*

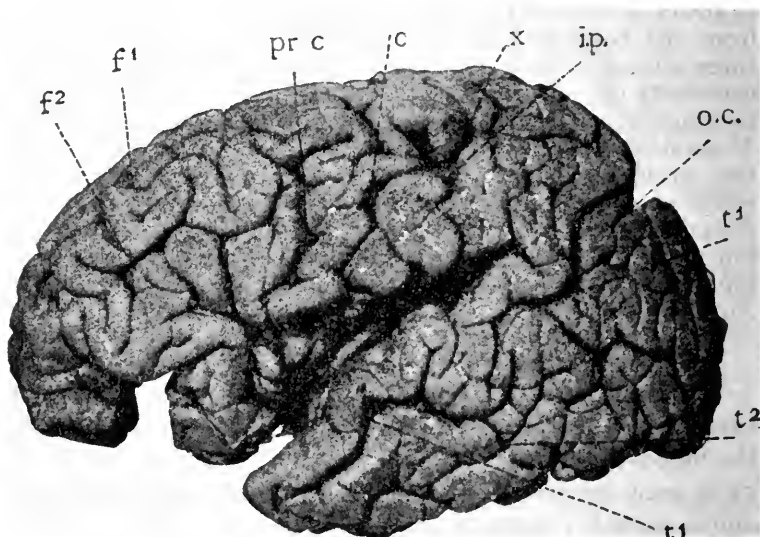
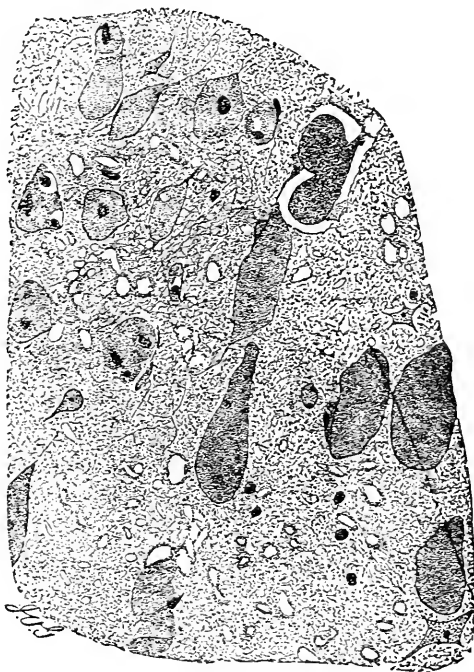


Fig. VIII.—Outer aspect of surface of left hemisphere, showing exposure of the island of Reil. (X) Region from which first block of tissue had been removed. (Cf. this journal, vol. x-14).

In the cases of Kast and Sachs there was no evidence of the agenesis having been due to early intra-uterine inflammation; in both cases there was a mere arrest of development and nothing more. The changes found in the case reported by one of us were limited to the large cells, in the cortex of which there was not a normal specimen to be found throughout the entire brain. There was no evidence of perivascular inflammation and only very slight thickening of neuroglia-tissue, with considerable retardation of development of the white fibres entering the gray matter of the cortex. The changes in the cells and the atrophies of the surface will be seen on inspection of the two accompanying figures. *Figs. VIII and IX.*

* A similar condition has been found by Jensen⁴¹ in a case of idiocy.

The pathology of paraplegia has not been determined. We have but one case with autopsy, that of Foerster,⁴² in which case Birch-Hirschfeld found a general cortical sclerosis with probable descending degeneration.* Ross was induced to state, only a few years ago, that he thought the



× 500 diameters.

Fig. IX.—Section of first temporal convolution. Distortion of pyramidal cells. Smaller cells and neuroglia cells distinctly nucleated.

changes of the spinal lateral columns due to traction at birth might be responsible for spastic paraplegia. This is a bare possibility; but the occurrence of idiocy and mental enfeeblement in 9 out of 11 cases of our own and in most of the reported cases, and the frequent occurrence of convulsions, make a cerebral origin much more probable.

The question arises whether it is possible to determine the morbid lesion in any given case, and we submit the

* See note page 301.

following as the result of our clinical and pathological studies :

The form of paralysis is not the most important factor in diagnosis.

Acquired cases of hemiplegia and diplegia, but particularly the former, are apt to be due to the same causes that prevail in adult apoplexies ; namely, hemorrhage, thrombosis, or embolism. Meningeal and cortical lesions are more frequent. Absence of convulsions at onset probably points to an intra-cerebral lesion. Occurrence of convulsions at onset points generally to cortical lesions or to embolism anywhere in the brain. In a few cases affections of gray matter lower down in the central nervous system may also be attended by convulsions.

Frequently repeated convulsions after onset point with great certainty to cortical lesion.

Cases coming on after acute infectious diseases have been proved to be due to vascular derangement, particularly to hemorrhage and embolism, but some cases may be due to a polioencephalitis corticalis ; the onset with very high fever and convulsions makes the latter more probable.

Traumatism is an important factor in the causation of meningeal hemorrhage during early life and particularly during the act of labor.

Congenital cases of diplegia and hemiplegia may be due to early meningeal hemorrhage and possibly to an early encephalitis.

In acquired and congenital cases of hemiplegia and diplegia we may have either a condition of porencephalus or simple arrest of development. If acquired, there would seem to be the history of slow development of the symptoms without predisposing cause. If congenital, there may be a history of traumatism to mother.

In later life hemiplegia or diplegia associated with large amount of mental impairment and with contractures point to general atrophy, sclerosis, with secondary degenerations, or to a condition of porencephalus, the origin of which it is often difficult to determine.

In conclusion, a word regarding treatment. So far as prophylactic measures are concerned, we wish to repeat our warning to the obstetrician: Hasten protracted labor, for the skillful use of forceps and careful manipulation are less apt to do injury than the prolonged compression of skull and brain in the pelvic canal. In the acute cases the same principles which obtain in the treatment of adult cases should be applied.

Medical advice is generally sought for the relief of the secondary conditions—epilepsy, idiocy, and contractures. In the treatment of the epilepsy the usual remedies may be employed: above all, the bromides and chloral. But these should be administered with the utmost discretion, for in many cases the epileptic attacks are so infrequent that they do far less harm to the patient than the drugs, which increase mental stupor and are very apt to cause anæmia and malnutrition. The proper use of electricity and massage in the early stages will prevent to some extent the formation of extreme contractures. If such contractures exist, we place our reliance upon the orthopedic surgeon, who finds in these cases a very wide field for the exercise of his surgical and mechanical skill. In cases in which the hand is so deformed as to be of less use than an artificial hand would be, amputation would be justified, provided there were no marked rigidity at the elbow. This has been thought of in the case of J. K., whose portrait has been shown. In one case, in this city, with extreme athetosis, the arm was amputated and the patient rendered more comfortable. This should be done only in the severest forms.

The question of surgical interference in cases of hemi-epilepsy deserves passing notice. In very recent cases, in which there is no prospect of spontaneous recovery and in which the character and site of the lesion can be accurately inferred, the idea of operation might be entertained, but it is wholly inadvisable in cases of long standing in which secondary conditions have been permanently established. The result in two cases of Bullard⁴³ and Mitchell⁴⁴ has not been encouraging.

Trepanation for the relief of severe headaches and local epilepsy may be attempted.

As for the condition of mental enfeeblement, much can be done by careful manual and mental training. This should be begun at an early day, and the results will be as satisfactory as in Bourneville's department of the Bicêtre, where the improvement is evident from the change in the facial expression of patients on admission and after they have been under treatment and instruction for some time. In only two cases that we have seen of infantile palsies can we conscientiously state that there has been complete recovery.

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THERAPEUTIC BREVITIES.

Under this title, the "Medical Age," Feb. 10, 1890, publishes some excellent gleanings. From the "British Med. Journal" is some consideration given to simulo in epilepsy, a remedy that Starr finds useful when the bromides fail. For infantile convulsions, Widerhofer, of Vienna, recommends the following as a sedative:—

Hydrate of chloral,	-	-	-	1 drachm.
Distilled water,	-	-	-	3 fluid ounces.
Syrup of bitter orange peel,	-	-	-	1 fluid ounce.

A teaspoonful every two hours. (*Révue général de Clinique et de Thérapentique.*)

For Asthma.

Hydrate of chloral,	-	-	-	30 grains.
Iodide of sodium,	-	-	-	22 grains.
Simple syrup,	-	-	-	4 ounces.

In an attack, give a tablespoonful every hour. (*Ibid.*)

THE RATIONAL TREATMENT OF SCIATICA.¹

By GRÆME M. HAMMOND, M.D.

FIFTY years ago sciatica was described under the heading of "rheumatism." In one of the leading textbooks of the day it was tersely mentioned as follows: "When rheumatism attacks the nerves of the leg it is called sciatica." The treatment recommended in the same volume was as limited as was the description of the disease, and consisted entirely in a recommendation of an anti-rheumatic diet and the internal administration of turpentine. Since then, however, sciatica has been studied in all of its forms, and the remedies which have been recommended for its cure are legion.

Before a disease can be scientifically treated, the pathological conditions which produce it must be thoroughly understood. A few words, therefore, on the pathology of sciatica will not be out of place in this paper.

Sciatica can properly be divided into two classes: one, in which the morbid changes take place primarily in the nerve itself; the other, where the disease begins primarily elsewhere and affects the nerve secondarily. The conditions which are supposed to induce sciatica by directly affecting the nerve are gout, rheumatism, syphilis, neuromata, traumatism, and cold; while the diseases to which sciatica is attributed secondarily are extra-pelvic and intra-pelvic tumors, including a distended rectum, and diseases of the bone, particularly hip-joint disease. Of all of these causes, exposure to cold probably produces more cases of sciatica than all the other causes combined.

It was formerly supposed that when the skin over the sciatic nerve was exposed to cold, and sciatica resulted, that the sciatica was due to neuralgia, that is, that the pain

¹ Read before the New York Neurological Society, April 1, 1890.

was produced by the irritation of central sensory cells. We know now, however, that this form of sciatica is a true neuritis. In mild cases, and probably in the initial stage of all cases, the inflammation is limited to the sheath of the nerve, the irritation of the delicate *nervi nervorum* accounting readily for the localized pain along the course of the nerve. In severe cases there is not only inflammation of the nerve-sheath, but there is also inflammation of the interstitial tissue, which, by its increase in volume, and consequent pressure upon the nerve-fibres, may induce atrophy and degeneration of the nerve and consequent atrophy and paralysis of many of the leg-muscles. There is also, in the majority of cases, an exudation of leucocytes between the nerve and its sheath, which, by distending the nerve-sheath, probably accounts for some of the pain.

In regard to the influence of gout, rheumatism, and syphilis as factors in the production of sciatica, I think there is considerable doubt. It is possible, in a very small percentage of cases, that these diseases may predispose the patient to sciatica, or may perhaps induce it primarily, but clinical evidence, at least in my experience, does not give much support to the rheumatic, gouty, or syphilitic origin of sciatica. Gowers² believes that both rheumatism and gout are "potent factors in the production of sciatica," but holds that "cases in which the syphilitic nature of the disease is certain are extremely rare." Anstie,³ on the other hand, remarks: "But so far from agreeing with those who think this [rheumatism] is a frequent case, my experience teaches me that it is quite exceptional; nor do I believe that the common opinion could ever have arisen had it not been for the rage that exists for connecting every disease with a special diathesis which the profession flatters itself that it understands." He is even more emphatic in his denunciation of gout as a cause of sciatica, and concludes with the remark that, in his experience, syphilis is but rarely concerned in producing it.

² Diseases of the Nervous System.

³ Neuralgia, etc.

My own clinical experience leads me to adopt Anstie's views. Rheumatism, gout, and syphilis are very common diseases in this country, and yet it is extremely rare to find an individual suffering from any one of them who also suffers from sciatica. As assistant to the Medical Clinic of Bellevue Hospital, I had the opportunity of seeing a great number of patients suffering from these diseases. In my clinic at the Metropolitan Throat Hospital, which I had for over two years, fully ninety per cent. of the patients suffered from rheumatism and gout. In the neurological department of the Post-Graduate School, with which I have been connected ever since the school was organized, I have had the opportunity of studying very many cases of sciatica; and my experience in this connection has shown that the vast majority of cases of sciatica have never suffered from rheumatism, gout, or syphilis, and that of the hundreds of cases of rheumatism, gout, and syphilis, a very infinitesimal proportion have even had sciatica. Another factor against the theory of rheumatism and gout causing sciatica is that anti-rheumatic and anti-goutic remedies, while they relieve the rheumatism and gout, fail utterly to improve the sciatica in the least. Again, no post-mortem evidences of gout or rheumatism can be found in the sciatic nerves after death.

It is very probable that both rheumatism and gout lower the tone of the system to such an extent as to render the patient more liable to an attack of sciatica than he otherwise would have been; but there is little or no evidence to show that either of these diseases directly produces sciatica, or neuritis in any other part of the body, by direct action.

Syphilis has been known, in rare instances, to cause sciatica, either by the pressure from gummata on the nerve-trunk or by causing inflammation in the nerve-sheath by the direct action of the syphilitic poison in the system. In regard to the latter, I am as skeptical as I am that the poisons of rheumatism and gout directly produce inflammation in the sheath or substance of the sciatic nerve.

Reports of cases of sciatica directly traceable to syphilis are uncommon. Only two such cases have come under my observation.

Neuromata, traumatism (which includes blows, falls, wounds, and muscular efforts), and intra-pelvic and extra-pelvic tumors, all produce sciatica by the irritation of pressure, which, if it is continued long enough, induces neuritis. Diseases of bones and joints cause sciatica by the extension of inflammation to the sciatic nerve.

It will therefore be understood, from the preceding remarks, that sciatica, no matter what its source of origin may be, is to be regarded as a neuritis, and is to be treated as such. Of course, if the neuritis has been induced by injury, by pressure, or by the extension of inflammation, it is absolutely necessary that these conditions should be removed; but by simply removing the original cause of the irritation, the pain is not always arrested. In the meantime the constant irritation of the sciatic nerve has resulted in a neuritis, which may remain long after the original source of irritation has been removed.

Considering, then, that we have to deal with an ordinary case of sciatica due to exposure to cold, or that we have successfully removed the original cause of the sciatica, and the pain still continues, what is the most rational plan of treatment to be adopted? Pathologically we have to deal with inflammation of the sheath of the nerve and perhaps of the nerve itself, and with a sero-fibrinous exudation, which is usually between the sheath and the nerve, but is sometimes in the substance of the nerve itself. Clinically we are confronted by pain, which may be slight or agonizing, continuous or only present on motion, and, in old cases, by a certain amount of atrophy of some of the muscles.

For the relief of pain the remedies used should vary with the extent of the suffering. In the most severe cases, where the suffering is intense, it is absolutely necessary to use morphine. When such is the case, it should be given hypodermically in doses amply sufficient to relieve all pain, and should be injected hypodermically, and not given by the mouth; the fluid should be injected as near the nerve as possible, as there is some reason to believe that morphine has a tendency to reduce the inflammation in a nerve when

brought in contact with it. In milder cases, phenacetin, in a single dose of fifteen grains, which can be repeated in an hour if necessary, will be found to fulfill all requirements. Antipyrine and antifibrin can be used in place of phenacetin if desired. I have never seen any benefit derived from the internal administration of aconitin, atropine, gelsemium, or turpentine, remedies which are claimed to be very useful in relieving the pain of sciatica.

To relieve the neuritis itself, I depend almost entirely upon rest, the application of cold, and the use of electricity.

In regard to the value of rest in the treatment of sciatica, there can be no doubt. Every time the leg is moved the functions of the sciatic nerve are called into play. It is well known that the use of nerves and muscles induces a temporary congestion of the parts used, which would only have a tendency to aggravate a condition of already existing inflammation. Now, by rest I do not mean simply forbidding a patient to walk about, or even confining him to his bed, but I mean absolute rest to the limb, which can only be obtained by putting the patient in bed and applying a suitable splint to the leg. The splint I always use is the old-fashioned long splint, reaching from the axilla to the sole of the foot. It should be attached to the body by means of a bandage, and in the same manner fastened to the leg from the ankle upward to a point just above the patella. This leaves the thigh and the sole of the foot uncovered, a proceeding which is necessary for the proper application of the cold and electricity. The idea of using a splint in cases of sciatica is not original with me, though perhaps the method of using it is. The splint was first advocated by Dr. S. Weir Mitchell several years ago, and is, I believe, still frequently used by him. It gives the leg absolute rest, and should be used in all severe cases. In very mild cases it is not necessary. About every fourth day it should be removed, and passive movements of the joints and slight manipulations of the muscles should be carefully made, after which the splint should be readjusted.

Cold is a most serviceable therapeutic agent. I am aware that refrigerating the skin over the course of the sciatic nerve with sprays of chloride of methyl, ether, and other agents which produce intense cold has been advocated and is frequently used. I have employed these remedies, and, after a careful trial of them, it does not seem to me that they are as efficacious as a more moderate degree of temperature continuously applied. It is my custom now to apply cold by means of ice-bags packed against the posterior surface of the thigh. This can readily be done with the splint on if it is adjusted in the manner just described. My reason for preferring this form of cold is that, it being continuous, it soon reaches the nerve, and materially aids in subduing the inflammation; as the cold is not intense, the skin is never frozen. My objection to the sprays of chloride of methyl, ether, and other freezing sprays is that the cold is so great that the skin soon freezes, and the application has to be discontinued before the beneficial results of the cold can be experienced by the inflamed nerve. This is particularly true of the chloride of methyl, which freezes the skin as soon as it comes in contact with it. It seems to me that where the chloride of methyl acts beneficially at all, it must do so as a counter-irritant, and not as a refrigerant. In my opinion the ether spray is far superior to it, as it is of a lesser degree of cold, and can therefore be applied for a much longer time; but neither of these agents can compare to the almost continuous application of the ice-bags.

Electricity, when properly applied, is one of the most useful and important remedies we possess for the treatment of sciatica, but when improperly used only serves to aggravate the disease and retard the recovery of the patient.

The faradaic current should not be used at all in acute sciatica. It is an irritating current, both to nerves and to muscles, and is therefore contra-indicated. After the neuritis has disappeared and the muscles have become flabby from disease, or in old cases, where the nerve has been damaged and atrophy of muscles has resulted, faradaic applications may be beneficial, but in acute sciatica it should never be used.

The galvanic current may be applied in two ways: as a continuous current, and as an interrupted current. There is the same objection to the interrupted galvanic current that there is to the faradaic—that is, that it is irritative. Both of these interrupted currents are antagonistic to the principle of absolute rest, which I believe to be so important a factor in the treatment of severe sciatica. The continuous galvanic current, on the other hand, is of great service. It allays pain, probably in part by the anæsthetic properties of its positive pole, probably in part by reducing the inflammation in the nerve. In what manner it relieves the neuritis is not known. It is claimed that it promotes the absorption of the serous exudation between the nerve and its sheath. However this may be, it unquestionably does relieve the patient, and in many instances no other remedy is necessary except rest. Its manner of application is as follows: The negative electrode should be about nine by four inches in size, and should be strapped to the sole of the foot by elastic bands. The positive electrode should be about five or six inches square, and should be applied over the gluteal region, over the point where the sciatic nerve emerges from the pelvis. If there are any very tender spots along the course of the nerve, this electrode can be changed occasionally so as to cover them. The strength of the current should not be such as to cause much pain, but should fall just short of doing so. No rule as to the current-strength to be employed can be laid down, as the point of toleration is different in different individuals. The continuous current should be applied twice daily, if possible, certainly once a day, for about five minutes at each *séance*. Most of the text-books recommend that at the end of each application of the continuous current a number of interruptions should be made in order to stimulate the muscles. Nothing of the sort should be done. It is opposed to the scientific treatment of the disease. It irritates the nerve, and counteracts, in part, if not altogether, the benefit derived from the continuous current.

As for the internal administration of drugs, there is very little to be said. In those cases which are unquestionably

syphilitic, of course anti-syphilitic treatment is indicated. In all other cases I think the iodide of potassium can be given, in gradually increasing doses, with great advantage, as it acts energetically in promoting the absorption of the serous exudation, and prevents, in a great measure, the formation of new connective-tissue.

Regarding sciatica from its pathological standpoint, it seems to me that the measures just alluded to—that is, absolute rest, the application of moderate but continuous cold, and the proper administration of the continuous galvanic current—constitute, with proper anodynes, to temporarily relieve pain, the rational and scientific treatment of the disease. In cases of moderate severity, rest, together with galvanism, will be the only remedies required.

In regard to other forms of treatment, a word must be said.

The use of colchicum, salicylic acid, salol, oil of winter-green, and other anti-goutic and anti-rheumatic remedies have not been followed by beneficial results in my cases, even where gout or rheumatism has complicated the case. Though the gout and rheumatism may yield to these drugs, the sciatica does not.

Blisters or the actual cautery are serviceable, but do not compare to the action of continuous cold. When the case is not a severe one, blisters or the cautery may be substituted for the cold.

Hypodermatic injections of various substances are frequently recommended as curing cases of sciatica. Among these may be mentioned ether, nitrate of silver, and osmic acid. Their action is so uncertain, and their tendency to create deep-seated abscesses is so well known, that I do not advocate their use.

The following cases, taken from a series of similar ones, will, I think, illustrate the points of the treatment just advocated :

CASE I.—A German, forty years of age, came to my clinic at this school in 1888. He had contracted sciatica in the left leg three weeks previously while standing at his

work while a strong draft was blowing on him. He was in great pain and walked with difficulty. There was no history of gout, rheumatism, or syphilis. I advised him to enter the hospital, but he refused to do so. I treated him as best I could for about four weeks, seeing him twice or three times a week. At the end of that time there was but slight improvement. He then entered Mt. Sinai Hospital, where he remained three weeks. While he was there he says "he took medicine, was told to keep quiet, and had a battery used on him five times." At the end of three weeks he reappeared at my clinic worse than he was before. He signified his willingness to enter the hospital; so I put him to bed at once, put the affected leg in a long splint (reaching from the axilla to the foot), packed ice-bags on the posterior surface of the thigh, gave orders that he was not to leave his bed, and had the continuous galvanic current applied, in the manner I have just mentioned, twice a day for five minutes at a time. In three days the pain had entirely ceased. I then left off the ice-bags; on the same day I removed the splint, and, after slight passive movements of the limb had been made, replaced it. This I did every three days. At the end of sixteen days the patient was discharged cured. I have seen him several times since then. There has been no recurrence of the attack.

CASE II.—A woman, forty-two years of age, with a decidedly rheumatic history. When I first saw her, on October 12, 1889, she had been suffering from a severe attack of sciatica in the right leg for over three weeks. It was not known how the attack originated, as there was no history of exposure to cold. Her family physician, believing it was of rheumatic origin, had treated her with salol and oil of wintergreen, but without materially lessening her sufferings. Morphine has been used to allay the pain. The treatment described in the history of the first case was carried out in this case, with the addition of the iodide of potassium in gradually increasing doses.

For the first three nights it was necessary to give morphine; but each night a reduced dose was given. On the fourth, fifth, and sixth nights the morphine was omitted, and ten grains of antipyrine were given at bed-time. After that no anodyne was used at all. There was a steady diminution of pain from the first. The splint was removed on the twelfth day, and the electricity was stopped on the eighteenth day, when she was pronounced well. She has had no return of the attack since.

CASE III.—A German woman, aged about fifty, consulted me, in November, 1888, for sciatica of the left leg. She had had it for about ten days. It began as a slight pain in the sciatic nerve, which had gradually increased, until at the time I saw her it was very severe. She was confined to bed, and was evidently in great pain. I ascertained that she had suffered from chronic constipation for several years, and that, for the past few months, five or six days would pass without there being a movement of the bowels. She would then take a strong cathartic and relieve herself for the time being. When I saw her she had not had a movement for five days. She has no history of gout, rheumatism, or syphilis. I directed my attention to relieving the bowels, supposing that when this was done the sciatica would disappear. It did disappear in a great measure, there being no pain at all as long as the patient remained quiet, but, almost as soon as she began to walk, the pain in the sciatic nerve would be felt, and would increase if exercise was persisted in. I confined her in bed for a week, not, however, using the splint or the ice-bags, and made applications of the continuous galvanic current twice daily. At the end of a week the patient could walk without the slightest pain.

These three cases, though taken from a large number, are not selected cases, but represent fairly the general average. In cases of long standing, where continued inflammation has produced organic changes in the nerve, with probable destruction of nerve-fibres, as shown by paralysis and atrophy of muscles, this form of treatment is not claimed to be efficacious.

“CEREBRAL SURGERY.”

From reports of the St. Louis Academy of Medicine, this department of surgery is not yielding very brilliant results. A patient with hemiplegia, aphasia, Jacksonian epilepsy, and otorrhœa was trephined on the supposition, that an abscess existed in the temporal lobe. The operation resulted fatally, and autopsy revealed three tumors in the central convolutions. Two other cases were reported where a correct diagnosis was made, but death followed the operation in each. (“Medical Record,” March 22, 1890).

Periscope.

By LOUISE FISKE-BRYSON, M. D.

CHOREA AMONG THE INSANE.

The "American Journal of the Medical Sciences" (April, 1890) contains a paper on this subject by Theodore Diller, M.D., in which appear the following conclusions:

I. There is to be found among the insane in hospitals and asylums in this country 1 choreic among each 425 of population.

II. In all long-standing cases of chorea there is more or less marked tendency to mental deterioration, which, in many cases, progressively increases, and finally terminates in dementia.

III. Many cases, even when there exists a considerable degree of mental impairment associated with chorea, enjoy fair physical health and apparently live almost as long as they would have done had they been free from the mental and nervous affection.

IV. The proportion of male to female adults is about the same ratio as is found to exist between the sexes in children affected with acute chorea.

V. The same causes that are known to produce chorea in children are found to operate in causing the disease in adults; but, in the case of the latter, additional causes peculiar to adult life, such as apoplexy, anxiety, etc., are capable of producing the disease.

VI. Persons of adult years are sometimes, though rarely, attacked while suffering from rheumatism—the disease being of about the same character as that commonly observed in children, but more likely to become chronic.

VII. As to pathology, the following appear to be reasonable conclusions: (a) A number of cases arise from, and are caused directly or remotely by, an attack of rheumatism. (b) In the majority of cases, heart-disease is absent, and there is a negative history as to rheumatism. (c) Coarse lesions, acting as irritants to the motor cells of the brain or the tracts proceeding therefrom, are in some cases the prime cause. Such lesions most commonly are clots recent, organized, or broken down.

VIII. Chorea is found at all ages.

IX. Persons may inherit the disease directly.

X. The disease may be congenital.

XI. Chorea and epilepsy are intimately related to each other. Epileptic convulsion (Jacksonian) may be confined to a single member; the same is true of choreic convulsions.

DUBOISIA SULPHATE IN PUERPERAL MANIA.

The "College and Clinical Record," January, 1890, states that Roberts Barthalow advises the subcutaneous injection of duboisia sulphate, gr. $\frac{1}{150}$ to $\frac{1}{60}$, in puerperal mania characterized by delusions and systematic depression. The indication for *atropia in epilepsy* is depression; in the opposite condition, the bromides are indicated.

MYXŒDEMA AND CO-EXISTING EXOPHTHALMIC GOITRE.

In a critical review by P. Kovaleski of "Myxœdema or the Pachydermic Cachexia," contained in the "Archives de Neurologie," November, 1889, there is given an interesting history of this unfortunate combination. The patient, an only child, forty-six years old, had the poorest kind of a chance, in point of heredity. Her paternal grandfather was a drunkard, and died in an insane asylum. Her father committed suicide at the age of twenty-five, a victim to melancholia. The mother was an epileptic. At fourteen the patient talked and walked in her sleep, though strong and well during childhood. The girl married at eighteen, very unhappily. Three or four attacks of epilepsy a year (*petit mal*) began now to manifest themselves. At twenty-four, one attack was followed by automatic acts, such as attending to household affairs, going out, making purchases, of which she had no recollection. Once or twice a year this happened; and when the woman was about thirty-two this psychic automatism became of longer duration, lasting eighteen hours, during which time she was irritable, quarrelsome and even violent. She had hallucinations, and moments of terrible anguish and anxiety. When forty years old, the patient left her husband, became poor and began to earn her own living. Periodic tachycardia now set in, and later became violent and continued. In two years, exophthalmia appeared; and eighteen months afterward, enlargement of the thyroid. She was sometimes better, sometimes worse, with frequent attacks of epileptic violence. During these attacks the symptoms of Basedow's disease were intensified, and became less pronounced when the seizures disappeared. The patient was carefully examined during a paroxysm of epileptic violence, and the following conditions noticed: feet and legs swollen, from the ankles up to the knee; skin stretched and dirty yellow in color, shining,

and denuded of hair, cold and dry to the touch; sebaceous and sudorific secretions absent. There was pitting or rather displacement on pressure, for the depression made by the finger disappeared when it was removed. The only heart symptom was acceleration of its beat (120-140). The urine was non-albuminous, of yellow tint, acid reaction, and containing a considerable quantity of urates (1015). Swelling similar to that in the feet existed in the cheeks and lips. The eyelids were much wrinkled, but not swollen. Hair on the head had become thin, and there was none whatever in the axilla. These symptoms, together with poverty of blood, insomnia, hallucinations of sight and hearing, manifestations of fear and terror, complete "absence," and a tendency to violence made up a picture of sufficient misery. This was succeeded by tranquillity in about three days. But her peaceful condition was not one of quiet, not post-epileptic depression. There was confusion of mind, indistinctness of speech, indifference, loss of facial expression, and general stupidity. Warm baths, galvanism (subaural), quinquina, small doses of arsenic, and somewhat energetic treatment, slowly brought about improvement. In three months all swelling had disappeared, and her psychic state became natural. What remained, however, were anæmic and exophthalmic goitre.

The patient stated that five or six months previous to this last attack she began to be so weak, broken, and exhausted, that her only desire was to stay constantly in bed. Thinking was difficult, or, to be more exact, she had no desire to think. Physically, she was not ill. The temperature was subnormal, and weariness so great that walking or working became almost impossible. She would sit down anywhere, thinking of nothing, and completely broken up. The swelling of the hands was not always present. Without known cause she lost eight teeth during the second month of her illness. Appetite and digestion remained good. There was no perspiration or oily secretion. The tongue became swollen, the saliva abundant, sticky, and thick. The patient always complained of feeling cold, especially in the parts that were swollen. There was but slight change in the thyroid gland at any time. It was a little larger during the epileptic delirium.

TROPHO-NEUROSIS AS A FACTOR IN THE PHENOMENA OF SYPHILIS.

The "St. Louis Courier of Medicine," Feb., 1890, calls attention to a paper with this title by Dr. G. Frank Lydston, in which the author affirms that syphilitic, as well as other

fevers, are due the action of a specific poison upon the sympathetic, and that the syphilitic poison may produce disturbance of the sympathetic with perversion of tissue, of metabolism, and excessive production of heat. The inconstancy of the syphilitic fever is explicable on the ground of idiosyncrasy. The roseola is due to vaso-motor changes with dilatation of the capillaries. In pronounced syphilitic lesions, the accumulation of cells is an exaggeration of normal tissue-building that is presided over by the sympathetic. Syphilitic infection has a peculiar affinity for the nervous system, especially for the upper and cervical portion of the sympathetic. The proportion of lesions of the head, face and mouth is larger than other portions of the body, especially those parts supplied by the fifth cranial nerve. The affinity of the specific infection for the iris may be easily explained in view of its sympathetic distribution. Syphilis seemingly possesses the power of dissecting out definite portions of osseous tissue (apparently by cutting off their nutrient supply) in a manner as clearly as it can be done by the knife. The explanation of destruction by pressure of syphilitic exudate will not suffice in these cases. Carefully observed, it will be found that the first symptoms experienced by the patient are those incidental to the presence of a foreign body, *i.e.*, dead bone in the tissues. If pressure were the cause of the necrosis, the death of the bone would be preceded by more or less painful swelling and inflammation. A perversion of trophic function in the nerve filaments supplying the parts is the only plausible explanation. All the pathological processes incidental to syphilis—whether the poison be microbe, degraded cell, or chronic abnormality—are due to disturbances of nutrition, resulting from the profound impressions made upon the sympathetic.

Asylum Notes.

BY FRANK H. INGRAM.

Dr. Charles W. Pilgrim, formerly an assistant at the Utica Asylum, has been appointed medical superintendent of Willard Asylum, to succeed Dr. P. M. Wise. Dr. Pilgrim, during his long experience with the insane, has gained an enviable reputation as a physician and an executive officer.

Dr. Geo. F. M. Bond resigned the superintendency of the Ward's Island Asylum on March 10th, to enter general practice in this city. Dr. Bond's successor, Dr. William

Austin Macy, was assistant superintendent, and his recently vacated position has been filled by the transfer of Dr. Lucius C. Adamson from the Blackwell's Island Asylum.

During the last few weeks the physicians at the Ward's Island Asylum have had served upon them numerous writs of *habeas corpus*. These writs were issued upon the application of a former asylum patient, who it would seem, has taken upon himself the task of attempting to liberate some dozens of men believed by him to be sane and unjustly confined. With a single exception, in which the jury's verdict "sane" was scarcely in keeping with the evidence presented, the patients were either remanded to the care of the asylum physicians or were discharged *in custody* of responsible friends.

Dr. Dwight R. Burrell, resident physician of Brigham Hall, Canandaigua, N. Y., and Miss Clara E. Kent, of Kentland, Ind., were married March 20th.

We regret to learn that Dr. J. S. Dorsett, superintendent of the State Lunatic Asylum, Austin, Tex., was attacked and dangerously injured by a patient named McDermott. Dr. Dorsett was struck on the back of the head with an iron bar, and the assault was the outcome of a delusion which originated in the mind of McDermott at the time of the hanging of murderer McCoy, against whom the doctor testified. This is another striking example of the dangers to which asylum physicians are particularly exposed. Since 1875 several men prominent in the profession—notably Dr. Cook, of Canandaigua, Dr. Sawyer, of Providence, and Dr. Metcalfe, of Kingston, Ontario—have died at the hands of insane patients; and the death of Dr. John P. Gray, of Utica, was, in great measure, due to the bullet-wound inflicted by an insane barber.

Dr. Samuel B. Lyon, for several years identified with the management of Bloomingdale Asylum, in the capacity of assistant medical superintendent, has been formally appointed medical superintendent, to fill the vacancy occasioned by the death of Dr. Charles H. Nichols. The promotion of Dr. Lyon is a most deserving one, reflecting credit upon the institution and its board of governors.

Dr. J. Elvin Courtney, formerly interne at Bloomingdale Asylum, has been appointed assistant physician to the Hudson River State Hospital, Poughkeepsie.

Dr. Charles Gray Wagner has been promoted to the position of first assistant physician in the Utica Asylum, to succeed Dr. Pilgrim.

Dr. Wm. D. Granger, for eight years first assistant physician at the State Asylum for the Insane, at Buffalo, N. Y. opens a private institution for the insane at Mt. Vernon, near this city, on June 1st. Dr. Granger has created for himself not only a State, but a national reputation, through the important part he took in aiding Dr. J. B. Andrews to make the Buffalo asylum the model institution of this country, and by the wide dissemination of his "Handbook for Training Nurses upon the Insane," published some years ago by G. P. Putnam's Sons. He has purchased the Masterson estate, midway between Mt. Vernon and Bronxville, consisting of an elegant stone mansion, cottages and seventeen acres of beautiful grounds upon the high ridge between the two towns. This institution will be the nearest to the city and most convenient of reach among those now in existence.

New Instruments.

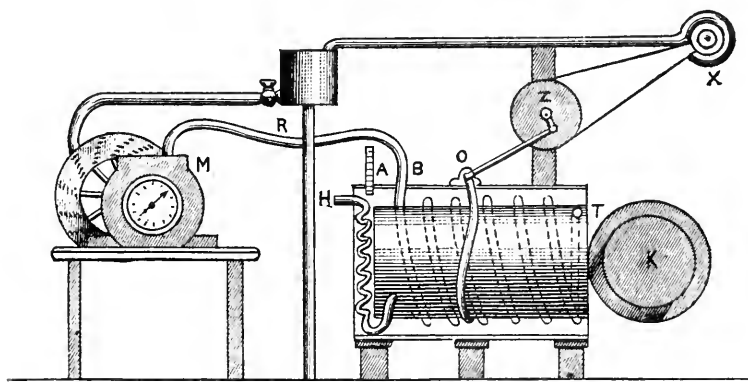
AN IMPROVED CALORIMETER.

By ISAAC OTT, M. D.

THE great importance of the study of the heat production and heat dissipation of animals has made it necessary to perfect the heat-measuring instrument, usually called a calorimeter. This instrument may be either an air or water calorimeter. Without going into any historical details, it is permissible to state that Rosenthal and Rubner have recently constructed two air calorimeters, which are held to be quite reliable. But as they require greater care in the management of them than water calorimeters, the latter have been extensively used. Of the water calorimeters, the old one of Despretz, and Du Long, and that of d'Arsonval, are the most important. In the Despretz and Du Long instrument the interior box of metal is submerged in the water, and through the water a serpentine coil runs to convey air to and from the inner box containing the animal. It is in the main the instrument that has been used in Germany and this country. D'Arsonval was the first to make the interior box immovable by fastening it to the outer wall of the water reservoir. He also made a door to close the opening into the animal chamber. It was an improvement upon the clumsy, time-wasting and inaccurate instrument of Du Long. It kept the temperature constant by means of the expansion and contraction of the mass of water regulating a gas burner. Whatever change of temperature in the animal must then be due to changes in the

tissues. It also had a serpentine coil for removing the air from the enclosure. I have used this calorimeter of d'Arsonval's, but in a manner not intended by its originator. The gas arrangement was dispensed with, and it was managed as other water calorimeters. Reichert has also constructed a calorimeter where the interior box is arranged in an immovable manner by attaching its inner reservoir to the outer wall of the water chamber.

That the animal may be quickly placed in the interior of the instrument he has inserted a plug into an opening of the instrument, in the place of a door. Otherwise it is essentially the instrument of Du Long. The error of Reichert's calorimeter is 8.75 per cent. In the improved calorimeter (Fig. 1) I have taken the cylinder form of d'Arsonval and made



several modifications. The agitator, *o*, sits astride the inner cylinder, outside the leaden coils, and is run at the rate of sixty to seventy times a minute by means of a water motor, *x*. In other calorimeters, the water is occasionally agitated by a hand contrivance. Instead of the air entering the inner chamber in a straight tube, it traverses the tube *H*, coiled upon itself in the water reservoir of the instrument, and enters the enclosure at its base. The air emerges at the opening *T* at the top, and is carried out through the serpentine coil and through the aspirating meter *M*, which records at the same time the amount of air. The constant activity of the agitator causes the water to equally diffuse the heat and to permit none to be given to the air. In the instrument of Reichet, nearly one-sixth of the heat is given to the air, and like all air calculations this is conducive to error. The door *K*, swings upon a hinge, and in its centre has a glass by which one can readily see the state of the animal or the apparatus connected with it. At its edge it is lined

with rubber and closed by powerful iron screw clamps. In front of the door is a mattress of saw-dust several inches thick. Over and around the calorimeter, instead of the usual saw-dust or felt, I used the packing material of wood fibre known as "Excelsior." The whole is enclosed in a square box, which has a door. The calorimeter is sixteen inches in length and twelve inches in diameter. At *A* is a circular opening, through which a thermometer, graduated to $\frac{1}{30}$ of a degree Fahrenheit, passes into the water and enables one to easily read a hundredths of a degree. At *B* is an opening in the air tube for the air thermometer to be pushed in.

The calorimeter is made up of the following parts: water, 30.72 lbs.; iron, 17 lbs.; solder, 1.50 lbs.; lead, 51 lbs. By multiplying these with their "specific heats," there is obtained the number 34.32.

		Sp. heat.		
Water, 30.72 lbs.	×	1.00	=	30.72
Iron, 17.00 "	×	.1138	=	1.9346
Solder, 1.50 "	×	.0789	=	0.1183
Lead, 51.00 "	×	.03050	=	1.5555

34.3284

34.32 = units of heat necessary to raise the calorimeter 1° F.

To test the instrument as to its accuracy, I used a tin vessel, closed on all sides except a small opening for the introduction of a thermometer and the heated water. This opening is closed by a metal screw-cap. When the weight and temperature of the water in this test-vessel are known, it is put in the calorimeter, and air aspirated through the calorimeter. Below is an experiment showing the method.

R. T. means	-	-	-	room temperature.
A. T. "	-	-	-	air "
C. T. "	-	-	-	calorimeter "
E. T. "	-	-	-	exit tube "
V. T. "	-	-	-	tin vessel "
M. "	-	-	-	meter "

TIME. P. M.	R. T.	A. T.	C. T.	E. T.	V. T.	M.
1.30	73.7		71.48		93.7	706447
1.40		73.4		22.2°C.		
1.50		73.7		22.3		
2.00		73.4		22.3		
2.10		73.3		22.4		
2.20		73.6		22.5		
2.30	73.3	73.3	72.46	25.6	83.2	711055

73.5	73.4	+ .98	22.3°C	10.5	4608
1.0 Correction	1.05 Correction		72.14°F.		Litres.
			.35 Correction		
72.5	72.35		71.79		
	71.78				
		.56 loss of temp. of air.			
Weight of tin vessel and water	= 4.42 lbs.				
Weight of tin vessel	= 1.3				
	Water = 3.12				
	Heat lost by vessel of water = 33.85				
	Heat accounted for by calorimeter = 32.02				
	Error = 5.4 %.				

As is seen by the calculations the error is 5.4 %. I performed a half dozen experiments, and found the variations from this number to be within a degree; hence, it is necessary to assume that it is an instrument of precision. For absolute accuracy the moisture of the air and the barometric correction should have been made, but they would not alter the result perceptibly. It was always used with the air a degree or so above the temperature of the calorimeter. The agitator was also set in motion for a half hour before the observation commenced. The room temperature for twenty-four hours previously was kept nearly the same. With these precautions the instrument works accurately. This instrument has been used during the past year in my laboratory.

Book Reviews.

A TEXT-BOOK OF MENTAL DISEASES, WITH SPECIAL REFERENCE TO THE PATHOLOGICAL ASPECTS OF INSANITY. By W. Bevan Lewis, L.R.C.P. Philadelphia: P. Blakiston, Son & Co., 1890.

We most cordially welcome Dr. Lewis' book. It treats of mental diseases from an elevated and new aspect, and bears throughout the stamp of research and originality; and it is of especial value, as it contains as well the product of years of practical experience.

It is refreshing to find no quarreling over classifications, definitions of insanity, clinical romancing, or medico-legal inanities. It begins by a careful detail of the anatomical structure of the nervous system, especial attention being given to the cerebral cortex and the nerve-cells.

Here and there throughout this first section the author directs the reader to the most important histological elements that play so important a role in the pathological changes of certain forms of insanity.

Then follows a clinical section, based upon the analysis of four thousand cases of insanity treated at the West Riding Asylum, of which Dr. Lewis is medical director.

This section is introduced by a psycho-physiological consideration of the states of depression and exaltation. When scientists treat upon psychological subjects they become imbued with an idea that it is necessary to be abstruse, to coin picture-words; simple English descriptive phraseology gives way to "intellectual potentials" without "definite cohesion."

The reviewer felt, while reading this section, that his education had been sadly neglected or his "automatic segregation" was out of gear. But he had a "vivid realization" that very often sense was lacking and good English abused.

There is a vast difference between this especial part and the rest of the work, as though written by some one else. It is unquestionably scientific, yet it is labored.

In the truly clinical and pathological sections his descriptions are simple, instructive, and very interesting. The metaphysical would be also, if the author had not felt compelled to have written for Spencerian minds.

It seems a duty for reviewers of books to find something to criticise. It is much easier, no doubt, to criticise psychological deductions than to create them, but it is not fair to stamp "text-book" to this part of the work. We claim there should have been a few preparatory chapters and simple definitions and illustrations scattered here and there, to make the reading easier and the interpretation plain.

The pathological is most instructive, and well worthy the attention of all scientists.

The special subject of the "scavenger cell," and its part in the history of lymphoidal connective-tissue formations, is well advanced.

The question, Does the book fill all the conditions of a text-book? is hardly to be answered affirmatively. It seems to the reviewer to be, instead, directed to the physiologist, psychologist, and alienist. It is more than a text-book to the pabulum-searching student or the hurrying physician.

It is certainly an admirable and instructive treatise on mental diseases.

THE NEUROSES OF THE GENITO-URINARY SYSTEM IN THE MALE, WITH STERILITY AND IMPOTENCE. By Dr. R. Ultzmann, Professor of Genito-Urinary Diseases in the University of Vienna. Translated by Gardner W. Allen, M.D., Surgeon in the Genito-Urinary Department, Boston Dispensary. Philadelphia and London: F. A. Davis.

This excellent little volume, one of the "Physician's and Student's Ready Reference Series," will be welcome, not only to

many an American practitioner, who in his pilgrimage to Vienna was privileged to listen to the eloquent discourses of the late Prof. Ultzmann, but will also, as we feel assured, meet with a grateful reception by those not thus favored.

Well versed in all branches of medicine, the author possessed the rare ability of interweaving clinical experiences with teachings gained from the study of microscopy, physiology, and pathology into a harmonious unity. Like other literary productions of Ultzmann, the present work, which consists of a compilation of the two monographs, "Ueber die Neuropathien des männlichen Geschlechtsapparates" and "Ueber Potentia generandi und Potentia coeundi," is eminently practical, yet scientific, and its style very fascinating.

The chapter on Impotence and Sterility is especially well written, and, as the author's name alone would be a guarantee, is free from all charlatanism, which, unfortunately, cannot be claimed for some recent publications by otherwise reputable authors. The translation is excellent. We can conscientiously recommend the volume to every student and practitioner in search of knowledge on the topics therein contained.

A. F. B.

SPINAL CONCUSSION: Surgically considered as a Cause of Spinal Injury, and neurologically restricted to a certain Symptom Group, for which is suggested the Designation *Erichsen's Disease*, as One Form of the Traumatic Neuroses. By S. V. Clevenger, M.D. With thirty wood engravings. 8vo, pp. iv. to 359. Philadelphia and London, 1889: F. A. Davis, Publisher.

This work, attractive in size, form, binding, and impression, is of great interest to the neurologist as well as to the general practitioner in localities in which accidents frequently occur. While those of the latter category whose scientific training is incomplete may find some of the technicalities of the book a little beyond them, they will still find in this most recent *exposé* of the subject so much that is helpful and even necessary to their success, that it becomes quasi-indispensable to them.

The author imparts much of his originality to the work by his independent appreciation of the good and also of the indifferent endeavors of other authors on spinal concussion. On more than one page he likewise justly deplores the insufficiency of medical education in our country. Those interested in the progress of that most subtle and difficult specialty, neurology, will read with discouragement and, at the same time, admit the truth of the following lines from page 25: "It is sad to reflect that the majority of medical men in our country have never seen a human spinal cord, would not recognize one if they did see it, nor would they know how to take it from its bony canal, and certainly would attempt to preserve

it in alcohol instead of Müller's fluid, and would be surprised to learn that alcohol would unfit it for microscopical examination."

After reviewing Erichsen on spinal concussion, and Page on injuries of spine and spinal cord, our author sums up recent discussions on spinal concussion, in which the most noted neurologists of the present epoch are cited, as Leyden, Westphal, Erb, Oppenheim, Spitzka, Putnam, Dana, Hammond, Seguin, Bramwell, Knapp, and others. This is followed by a chapter on Oppenheim's important work on "Traumatic Neuroses."

The chapter on the spinal column contains a summary of our latest knowledge of the anatomy and physiology of the spinal cord, and is accompanied by numerous plain and colored diagrams of great use in the study of this part of the body. It also contains many aphorisms of the highest importance pertaining to the pathology of the cord and of its membranes.

The most practical part of the work resides in the four excellent chapters on the symptoms of Erichsen's disease (spinal concussion), on diagnosis, on electro diagnosis, and on differential diagnosis. They are elaborated in a masterly manner, and are full of the most necessary data for the practitioner, be he specialist or not.

The most peculiar feature of the book is found in the chapter called "Pathology," but which should more appropriately be termed "Pathogeny," since it seeks to explain the cause and origin of the disease. Our author here develops the views, original with himself and "not heretofore advanced," which impute to injuries of the spinal sympathetic nervous system the preponderating rôle in the production of the varied symptoms embraced under the head of spinal concussion. That the sympathetic nervous system reacts upon the cord through vaso-motor nerves is not to be doubted and it is easy to admit that functional disturbance of the cord could result from traumatism of certain parts of that system. We are quite prepared to accept most of the propositions he sets forth in this chapter, supported as they are by experiments and opinions of numerous authorities in neurology.

A few directions concerning treatment and some excellent medico-legal considerations form the conclusion of this very recommendable treatise, of which the many good points cause us to overlook the fact that some of its phrases are not specimens of the most classical English.

W. W. S.

BOOKS RECEIVED.

INJURIES AND DISEASES OF NERVES, AND THEIR SURGICAL TREATMENT. By Anthony A. Bowlby, F.R.C.S. Philadelphia: P. Blakiston, Son & Co., Publishers.

A NEW MEDICAL DICTIONARY including all the words and phrases used in medicine with their proper pronunciation and definition. By George M. Gould, B.A., M.D.; P. Blakiston, Son & Co., Philadelphia: 1890.

PAMPHLETS, ETC., RECEIVED.

SANITARY ENTOMBMENT: THE IDEAL DISPOSITION OF THE DEAD. By the Rev. Chas. R. Treat. Reprint.

THE VAGUS TREATMENT OF CHOLERA, as exemplified in Returns from the Cholera Hospitals of Malta during the Epidemic of 1887. By Alexander Hackin, M.D., F.R.C.S. Reprint.

MULTIPLE NEURITIS. By Irving D. Wiltout, M.D. Reprint.

BRAIN AND SPINAL SURGERY IN PHILADELPHIA. By Irving D. Wiltout, M.D. Reprint.

AN EXPERIMENTAL STUDY OF LESIONS ARISING FROM SEVERE CONCUSSIONS. By B. A. Watson, A.M., M.D. Philadelphia, 1890: P. Blakiston, Son & Co., Publishers.

FIRST ANNUAL REPORT ST. BARTHOLOMEW'S HOSPITAL AND DISPENSARY. 84 Carmine St., New York.

TOBACCO AMBLYOPIA. By Leartus Connor, A.M., M.D. Reprint.

SKETCH OF THE LATE DR. J. EDWARD TURNER, THE FOUNDER OF INEBRIATE ASYLUMS. By Dr. T. D. Crothers. Reprint.

Miscellany.

SULPHONAL AND CHLORALAMID.

Daniel R. Brower, M.D. ("Medical News," April 19, 1890): "The medical profession to-day is more fortunate than ever before in the number of sleep-producing remedies. Sulphonal is a comparatively recent remedy of great value. It does not interfere with the digestion, the circulation, or the heart's action, as chloral, the bromides, and opium frequently do. We have also the still more recent remedy chloralamid. It has some advantage over sulphonal in that it acts more rapidly, and when dissolved in wine has but little taste. Sulphonal, on the contrary, probably by reason of its great insolubility, often acts slowly, its effects being more manifest the next day than on the evening of its administration. You can often administer either of these remedies without the patient's knowledge, as they have but little taste."

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It must be admitted, however, that many cases require frequently in devitalizing diseases some efficient method of rapid nutrition, capable of ready absorption without taxing the digestive functions, to combat the anæmia.

This is furnished most naturally by the circulating medium itself—blood containing the elements of nutrition in assimilable form—and a preparation of bullock's blood, entitled Hæmoglobin Compound, has been prepared, and is now marketed by Parke, Davis & Co.

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The Law Telephone Company have recently shipped eight of the "Law Galvanic Outfits" to Europe: three outfits of 60 cells each going to Paris; one to Boulogne; one to Berlin; two to London; and the last to Dr. Lawson Tait, Birmingham.

NEW WORDS.

We have been invited by a large publishing-house, who are preparing to issue a Complete Dictionary of the English Language, to send in a list of new words that ought to, in our estimation, appear in it.

Any words sent us will be gladly incorporated in a list to be sent them, and invite our readers to interest themselves in this important matter.

NOTICE.

They who read medical journals to the exclusion of the advertisements make a serious practical mistake. It is seldom that their perusal does not prompt the use of some forgotten remedy of value, reveals an address mislaid, or the superiority of some instrument.

The journal's advertisements ARE GREAT REMINDERS.

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It is most urgently requested that the personal friends of this journal will aid its advertisers and endeavor to make them feel that their support is appreciated.

WANTED.—Complete file of JOURNAL OF NERVOUS AND MENTAL DISEASE, bound or unbound, to 1888. Special numbers wanted: January, 1888; March, 1889.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

THE MINUTE STRUCTURE OF THE GRAY
NERVE-TISSUE.

By C. HEITZMANN, M.D.

IN the year 1873¹ I made the following statements with regard to the structure-elements of the nervous system: Thin sections from the cortex or the main ganglia of a recently killed grown rabbit are the best specimens for examination with high powers of the microscope. The section may be transferred to the slide with or without the addition of a preserving fluid; in the former instance a very dilute solution of bichromate of potash is preferable, because, as proved by A. Rollet, this does not alter the structure of protoplasm. Layers of protoplasm, with numerous formations like nuclei, ganglion-corpuscles of varying shapes, and medullated nerve-fibres of different sizes are seen. The living matter in the formations termed nucleoli, being compactly accumulated, is homogeneous and has a yellowish lustre; while in the protoplasma of all structure-elements of the nervous system the living matter is distributed in thin layers in the shape of granules and lumps, and is of an opaque gray color. All granules and lumps of the living matter are interconnected by means of delicate radiating spokes.

¹ Untersuchungen über das Protoplasma. Sitzungsber. d. Akad. d. Wissens in Wien.

In that article I first announced that all constituent elements of the gray nerve-tissue are constructed like protoplasm in general, viz., of a reticulum of living matter, the meshes of which contain a lifeless nitrogenous liquid. My illustrations plainly show this reticulum, both in the scattered nuclei, in the ganglionic corpuscles, and in the gray substance at large, the reticulum of the latter being uninterruptedly connected with that of the former.

The first observer, who maintained the presence of a delicate reticulum throughout the gray substance, more especially that of the spinal cord, was Gerlach, in 1870.² Since he was able to trace the filaments of this reticulum to the ganglion-cells, nay, their direct transition into the bodies of these corpuscles, he, apparently, was right in claiming that the reticulum itself was nerve-tissue. This assertion, however, was contradictory to the well-established doctrine in physiology that all nerve-conduction is insulated. How could there be any insulation of nerve-impulses, if these were to run through a nervous network pervading the gray substance as a whole? While Gerlach's observation was admitted to be correct, his claim of the nervous nature of the delicate network has been doubted and overthrown by many excellent observers. Golgi, in 1873³ and 1886,⁴ asserted that he was able to trace the filaments of the network to the ganglionic as well as to the connective tissue or glia-corpuscles, and, consequently, admitted the correctness of Gerlach's hypothesis in its essential features. At the same time he endeavors to draw a distinction between a purely nervous and a purely connective-tissue reticulum, denying the nervous nature of the broad or Deiters' offshoots.

S. Stricker, in 1883,⁵ thoroughly discusses the nature of the reticulum under question, pointing also to the broad offshoots of the ganglion-corpuscles, which were discovered by O. Deiters (1865), and termed by him "protoplasmic

² Art. Rückenmark. Stricker's Handbuch d. Lehre von den Geweben.

³ Tulla struttura d. sostan. grigia. *Communic. prev.* Milano.

⁴ Tulla fina anatomia degli org. centrali d. sistema nervoso. Milano.

⁵ Vorlesungen über allg. u. exper. Pathologie. Wien.

offshoots," the nature of which has remained entirely unsettled, since they could not be proved to become nerves, or axis-cylinders proper. Have we any right, Stricker argues, to call offshoots nervous, simply because they are in union

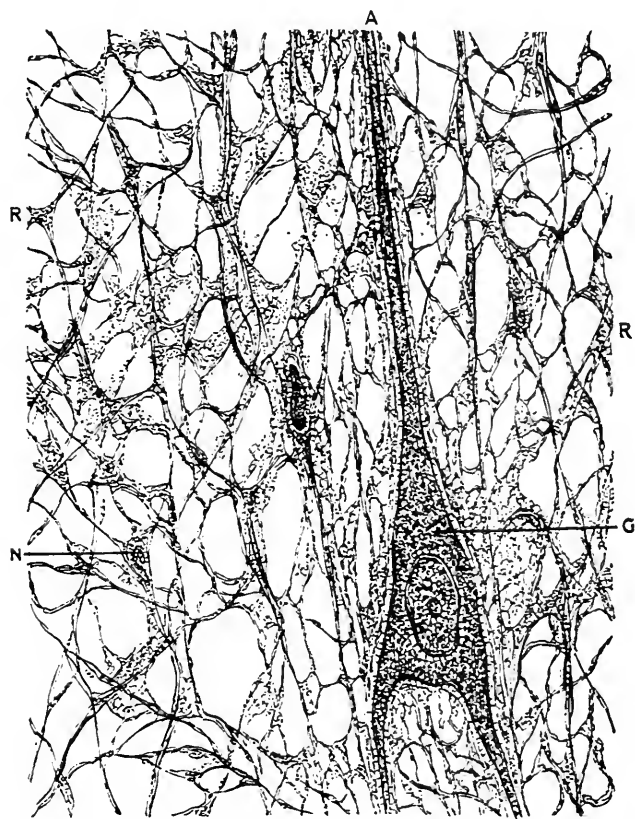


FIG. I.—Gray Nerve-tissue of the Spinal Cord of a Rabbit. Magn. = 1200.
G Tripolar ganglionic corpuscle. *A* Axis-cylinder offshoot. *RR* Reticulum of living matter. *N* Nucleus forming point of intersection in the reticulum.

with ganglion-corpuscles? A similar reticulum exists in the outermost layer of the cortex of the brain, which lacks ganglion-corpuscles altogether, whose nervous nature had already been doubted by Meynert, and which nevertheless is continuous with the network of the deeper layers, so profusely supplied with ganglion-corpuscles. According

to Stricker, the meshes of the network are filled with a finely granular substance, rendered conspicuous by certain reagents to such an extent that the reticulum itself becomes concealed by it. This filling may furnish the gray substance with its peculiar consistency. Stricker considers the whole gray substance as inert, kindred to connective tissue, and considers nerve-action as existing only in the ganglion-corpuscles.

A. Forel,⁶ while admitting the correctness of Golgi's assertions, disagrees with this observer in regard to the central sensitive corpuscles. He says that all the sensitive nerves terminate in the reticulum of the gray substance without special centres. Kölliker (1887) is opposed to the assertion of Golgi, that the protoplasmic offshoots of the ganglion-corpuscles are not nervous in nature.

I have thus briefly given the most important literature on this topic in order to show that the minds of histologists are still unsettled concerning the nature of the reticulum, which so profusely traverses and builds up the gray substance. Not only do some observers deny it all activity, but Kühne and Ewald (1877) have even demonstrated a good deal of the reticulum to be keratoid, horny, not being digestible in pepsin and trypsin.

Fig 1 gives an accurate representation of the morphology of the reticulum. The drawing is made from a specimen of the spinal cord of a rabbit, which was treated with osmic acid, cut with the microtome, and then stained with alumcarmine. We see that the reticulum in some portions is extremely minute, scarcely discernible with the excellent immersion-lens of Tolles, at my disposal, while in other portions it encloses somewhat larger meshes, which, to a certain extent, may have been produced artificially by the cutting and mounting procedures. The reticulum is continuous with that of the ganglionic corpuscle and that of the axis-cylinders. We can trace it to the walls of the capillary blood-vessels, where it traverses the narrow, peri-

⁶ Einige hirnanatom. Betrachtungen u. Ergebnisse. Arch. f. Psychiatrie, 1889.

vascular space, the same as it traverses the periganglionic space.

According to my views the gray substance is constructed in the same manner as protoplasm in general, *i. e.*, by a reticulum of living or contractile matter and a liquid filling the meshes of the reticulum. The structure of the gray substance is identical with that of the ganglionic corpuscles and the scattered, apparently isolated nuclei: the only difference being that in the gray substance the reticulum of the living matter is extremely thin and delicate; in the central form-elements, on the contrary, comparatively coarse and dense. Besides, there may be a difference in the chemical constitution of the filling liquid, which difference, however, is not perceptible to the microscopist. We have neither any right to call the reticulum nervous, in the sense of Gerlach, nor inert or connective tissue, as claimed by other authors.

In looking over the main varieties of connective tissue, *i. e.*, the myxomatous, the fibrous, the cartilaginous, and the bony, we find as the most characteristic feature the so-called basis-substance. This is greatly at variance in its chemical constitution, even in apparently kindred forms of connective tissue. In one essential point, however, it is alike in all forms, *viz.*, in filling the meshes of the reticulum and rendering it more or less consistent. The reticulum is concealed by the basis substance, and even in the myxomatous tissue never as plain as it appears in the gray nerve-tissue. It may be rendered conspicuous by different reagents, such as absolute alcohol, osmic acid, chloride of gold, etc. In the gray nerve-tissue such a basis-substance, the most important feature of connective tissue, is present only at the boundary-zone between the white and the gray substance (see Fig. 2, *g*), and in scanty bundles of a delicate fibrous connective tissue, which penetrate the gray from the white substance and run a radiating course through the former. At the boundary-zone the broad bundles of the interstitial inner perineurium rapidly decrease in bulk, for a short distance retaining a striated or fibrous structure, and soon splitting up into fibres which assist in building up the

reticulum. As long as the striated structure of bundles is recognizable, we have no reason to doubt the connective-tissue nature of the network. As soon, however, as the basis-substance proper is lost, the reticulum of living matter, traversing the basis-substance, is freed. Single granular fibrillæ, ever so conspicuously arising from the trabeculæ of the perineurium, are certainly not entitled to the name of connective tissue. Some of them may have undergone peculiar chemical changes (according to Kühne and Ewald, keratoid or horny), and thus serve as a supporting apparatus for the delicate reticulum; but they have ceased to be connective tissue. Where one ends and the other begins, the morphologist will never be able to tell.

Scanty bundles of fibrous connective tissue emanate from the pia-mater offshoots, which are likewise instrumental in constructing the inner prineurium and traverse the gray substance, as before mentioned, in a radiating direction. Probably they inosculate with the connective-tissue layer surrounding the ciliated epithelia of the central canal, although from personal observation I am unable to make such a statement positively. These bundles, being pierced by a reticulum of living matter, as all varieties of connective tissue are, profusely connect with the reticulum of the gray tissue, and again we are at a loss to tell at which point the basis-substance ceases and the living network has become free.

Unless chemical micro-reagents will be found, far more delicate than are at our disposal at the present time, I consider the task, to accurately discriminate in the gray nerve-tissue between fibrous connective tissue and fibrillæ of living matter, a hopeless one. What Stricker has claimed to be a granular filling mass is nothing but the most delicate portion of the reticulum itself, as evinced by the study of thin slabs with good immersion-lenses.

The reticulum, certainly in its main bulk, is alive during the life of the organism, and by its contractions causes that which we call nervous impulse. The reticulum of the gray substance is able to conduct in essentially the same manner, as that of the ganglionic corpuscles, by contraction, which

means narrowing of the reticulum and expansion, viz., widening of the reticulum. The effect will, however, be different in the widespread, loosely arranged, and extremely delicate network of the gray tissue and the dense, compact one of the ganglionic corpuscles. Both being identical in their nature, the final result may depend merely on the anatomical differences in the distribution and compactness. Any living lump of protoplasm is, as is acknowledged to day, movable and sensitive without a trace of nerves. The *amœba* creeps and evades obstacles in its way, it perceives the light, though it is built up by nothing but a reticulum of living matter, without differentiation into nerves or nerve-centres. Looking at the structure of the gray substance in this light, all difficulties in explaining its histological nature will fade away, and nerve-action becomes explicable. The absolute insulation of nerve-impulse is, consequently, done away with, and numerous physiological and pathological observations strongly point toward the absence of a perfect insulation within the nerve-centres.

The second topic under consideration is the structure of the axis-cylinder. This term includes the non-medullated nerve-fibres as well as the initial portions of the medullated nerve-fibres as yet destitute of a medullary investment.

Max Schultze, in 1868,⁷ was the first to assert that the axis-cylinders have, in many instances, a fibrillated structure which he claimed to be continuous with the fibrillated structure within the ganglionic corpuscles. S. Stricker, in 1883,⁸ admits that he saw the convincing specimens of Max Schultze, but at the same time declares that he was never able to see the fibrillated structure of the axis-cylinders in specimens of brains that were brought into hardening liquids immediately after the death of the animal. What the structure of the axis-cylinder is, this author does not say. I made the following statement in 1883:⁹ "With high am-

⁷ Die Structurelemente des Nervensystems. Handbuch d. Lehre von den Geweben, von S. Stricker.

⁸ Vorlesungen über allg. u. exper. Pathologie, p. 576.

⁹ Microscopical Morphology, p. 298. New York.

plifications of the microscope, some of the larger non-medullated nerve-fibres distinctly show a delicate reticular structure; others exhibit a number of minute vacuoles in their interior; still others, and these are the finest nerve-fibres, have a homogeneous appearance and give no evidence of structure." On page 309 I publish a diagram of nerve-conduction, in which the axis-cylinders, as well as the ganglionic corpuscles themselves, are drawn reticular throughout. Should A. Forel's above-quoted views be correct, the presence of a sensitive nerve-ganglion will prove to be a fallacy, but would have to be replaced by a reticulum of living matter within the gray substance, into which also the broad or Deiters' offshoot ought to inoscillate. That much plainly follows from my diagram, that I consider the structure of the axis-cylinder identical with that of the ganglionic corpuscles, viz., made up of a reticulum of living or contractile matter, much more delicate and dense in the axis-cylinders than in the ganglionic corpuscles. In the latter elements this structure is scarcely a subject of doubt, and since it was first discovered by C. Frommann (in 1867) has oftentimes been described and illustrated by excellent histologists; whereas the reticular structure of the axis-cylinder, at least as far as I know, was corroborated only quite recently by Max Joseph (1888).¹⁰ This observer has investigated the electric nerve-fibres of torpedo marm. He denies the keratoid or horny nature of the network within the myelin investment of the medullated nerves, since, contrary to the claim of Kühne-Ewald, it is dissolved by pepsin and trypsin, and demonstrates the presence of a much more delicate network in the axis-cylinder.

The question now arises: How was it possible that Max Schultze, one of our most skilled observers in microscopy, could have seen a fibrillated structure in the axis-cylinders? For nobody will doubt that such a structure was visible, if so asserted by M. Schultze. There is but one explanation to this strange fact, viz., the faulty method of teasing nerve-

¹⁰ Berliner Akademie-Bericht, p. 1321. Quoted from Edw. Klebs, Allg. Pathologie, II. Theil, 1889.

specimens and their mounting in Canada balsam. We can realize that by dragging delicate structures, such as those of ganglion-corpuscles and axis-cylinders are, the reticulum will be drawn out and be artificially transformed into a series of fibrillæ. At first teasing was done by M. Schulze in an indifferent liquid, the iodine-serum, and afterward, for fixation, alcohol and osmic-acid solution were resorted to. Fortunately, nowadays, all mutilating methods, such as

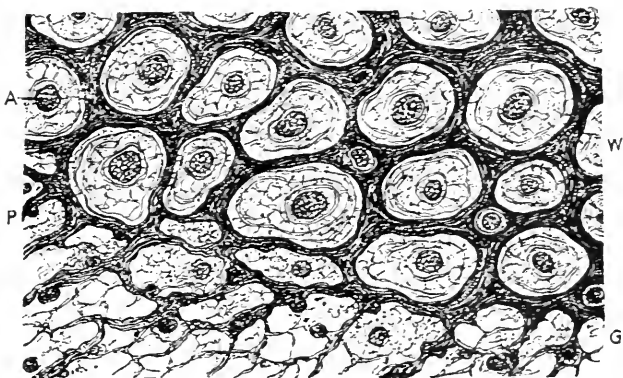


FIG. II.—Boundary zone between the Gray and White Nerve-Tissue. Spinal Cord of a Rabbit. Transverse section. Magn. = 1200. *G* Gray nerve-tissue. *W* White nerve-tissue. *A* Axis-cylinder. *P* Perineurium.

tearing and teasing unquestionably are, have fallen into discredit, and find application only by narrow-minded histologists, who are unable to appreciate the sad consequences of mechanical injuries and of laceration of tissues, the structure of which becomes comprehensible only by a strict conservation of integrity and continuity.

Since 1873 I have been convinced of the reticular structure of all nerve-elements to such an extent that I considered the ultimate axis-fibrillæ—composed of granules and a single inter-connecting thread, rendering these fibres rosary-like—merely a linear projection of the reticulum, inosculating with the living matter, both at the periphery and in the central gray tissue. To-day I have no reason to change my views, after having studied a number of specimens,

treated with widely different methods. Fig. 1 shows the delicate reticular structure in the axis-cylinder running longitudinally; Fig. 2, the transverse sections of these formations within the white mantel of the spinal cord.

Fig. 2 represents the boundary-zone between the white and gray substance, in the transverse section of the spinal cord of a rabbit. The specimen was treated first with osmic acid and afterward with alum-carmin solution. It is striking that the osmic acid did not stain the myeline in a dark-brown color, but the interstitial inner perineurium, the transition of which into the reticulum of the gray tissue is well shown. The axis-cylinders plainly exhibit a finely reticular structure. Around them we recognize a delicate sheath, the axis-cylinder sheath of L. Mauthner, and at the periphery of the fibre the sheath of Schwann, enclosing the myeline. Besides, here and there are visible other concentrically arranged layers, the significance of which I am unable to tell. The space, previously occupied by the myeline, is traversed by a knotty, irregular network, first described by Kühne and Ewald in 1886,¹¹ and claimed to be keratoid or horny by these observers, owing to the fact that it remains undigested under the influence of pepsin and trypsin.

In the finest axis-cylinders I am unable to discover any structure whatever, which does not exclude the possibility of a structure becoming recognizable, at some future time, by improved optical or staining appliances.

The presence of a contractile reticulum within the axis-cylinder would enable us to endeavor reducing nervous action to contractility, a feature found in all protoplasmic formations. The denser and more delicate the reticulum of living matter, the more rapid will be its contraction, furnishing the physiological basis of all nervous impulse. Centripetal contraction will be felt as sensation; centrifugal will result in motion. The nerves, when viewed in this light, merely convey rapid contractions. They may be considered as an apparatus of refinement of physiological

¹¹ Verhandl. d. Heidelberger Gesellsch.

properties, common to every living lump of protoplasm, animal as well as vegetable, *i. e.*, sensation and motion.

The third and last topic of my paper is the origin of axis-cylinders, or nerve-fibres, from the reticulum of the gray substance.

O. Deiters, in 1865,¹² made the assertion that from a ganglionic corpuscle, the number of its offshoots being ever so large, probably but one offshoot arises which is a true axis-cylinder, and which, coursing toward the periphery, respectively the white substance of the brain and spinal cord, becomes a nerve proper. Since that time this fact is generally admitted as correct. Deiters called all the other offshoots protoplasmic or branching. J. Gerlach, the discoverer of the nervous reticulum in the gray substance, in 1870¹³ first made the statement that Deiters' offshoots inosculate with the nerve-reticulum, and true axis-cylinders originate from this reticulum, not being in direct union with any ganglionic corpuscle. His description of the method best suited for the demonstration of this fact is briefly as follows: "A perfectly fresh, yet warm spinal cord of a calf or ox is sliced with the razor into very thin longitudinal sections, best through the anterior horns, and immediately placed into a very dilute solution of bichromate of ammonia (1 to 5,000 or 10,000 water) for two or three days. After this the slabs are placed into dilute ammoniacal carmine, and, after twenty-four hours, are torn up by means of needles, with special care for the preservation of the dark-red nerve-cells. Such specimens are preserved in glycerin, or, preferably, allowed to dry, and, after addition of a minute quantity of oil of cloves, mounted in Canada balsam."

I have purposely quoted Gerlach's words, in order to show that his assertions were based upon the study of teased specimens, as is also illustrated by his Fig. 223, the object of which is to demonstrate the origin of a branching nerve-fibre from the nerve-reticulum. No wonder,

¹² Untersuchungen über Gehirn u. Rückenmark.

¹³ Art. Rückenmark. Handbuch der Lehre von den Geweben, von S. Stricker.

therefore, that but little attention was paid to Gerlach's statement, which, nevertheless, is perfectly true.

In my "Microscopical Morphology" (1883), on page 288, I quote Gerlach's discovery, as follows: "The offshoots of the ganglionic elements are of two kinds: the broad, so-called protoplasmic offshoots of Deiters, and the narrow,

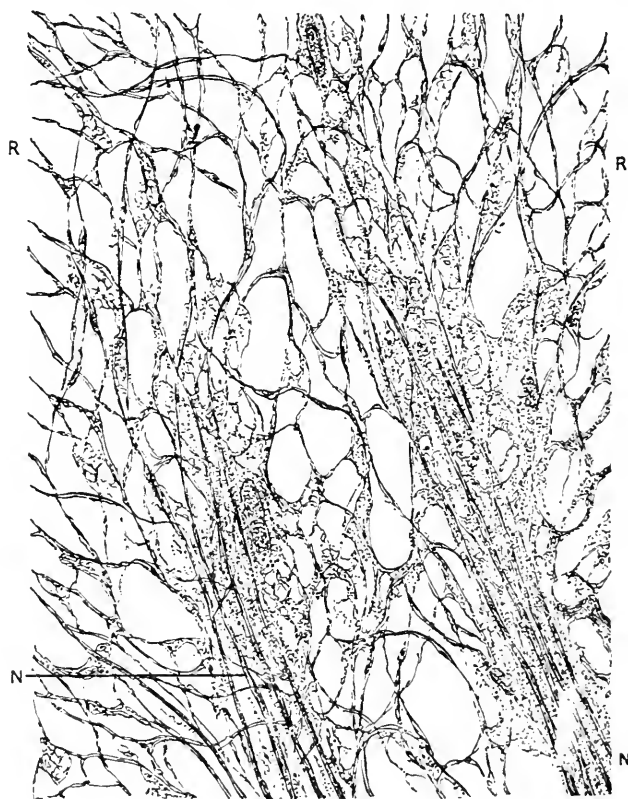


FIG. III.—Origin of Nerve-Fibres from the reticulum of living matter. Gray Nerve-tissue of the Spinal Cord of a Rabbit. Magn. = 1200. *NN* Bundles of nerve-fibres. *RR* Reticulum of living matter.

axis-cylinder offshoots. Of the former we know that they connect neighboring elements, and branch out into the gray substance, where they divide into an extremely delicate reticulum, first described by J. Gerlach. This author further asserts that the ganglionic elements of Clarke's columns,

and perhaps those of the posterior horns also, have no other than branching offshoots." On page 290: "According to Gerlach, it is also probable that from the reticulum of the posterior horn nerve-fibres originate, which in this horn and in the white substance take a centripetal course." On page 288 I make the following assertion, independently of Gerlach and based upon observation of sections of the spinal cord, carefully preserved in their continuity: "Axis-cylinder offshoots arise also from the gray substance, without any connection with ganglionic elements." This statement is a corroboration of that of Gerlach.

In order to show how such a statement could be made from the study of a sliced specimen, I affix Fig. 3, obtained from a spinal cord first treated with a one-per-cent. osmic-acid solution and afterward with alum-carmine.

We see the reticulum—the region is that of the anterior commissure branching into the gray tissue of the anterior horn—condensed into filaments, which, gradually assuming an increasing diameter, are traceable a great distance into the gray tissue, and have no other connections with the adjacent reticulum but the delicate lateral spokes visible on axis-cylinders generally, including those that plainly arise from a ganglionic corpuscle. Can there be any doubt as to the nervous nature of such filaments? I should think not.

In the posterior horns there are no ganglionic corpuscles proper; only formations like nuclei, which, in my conviction, serve as central formations, since in the brain of the lowest vertebrates they are the only elements visible in the gray tissue. My teacher, E. Brücke, often asserted that nobody has as yet been able to see nerve-fibres emanate from such nuclei in the posterior horns, and Gerlach's statement, that all sensitive nerves of the posterior horn inosculate with the reticulum of the gray tissue, is certainly correct. In 1883, while in Paris, L. Ranvier, the ablest French histologist, was kind enough to show me what he considered glia-cells, therefore connective-tissue elements, freely branching into innumerable offshoots and producing around the "cell" a delicate network. Golgi (*loc. cit.*) made at-

tempts to discriminate between a nervous and a connective-tissue reticulum, but failed to convince the histologists, since the differences in the taking up of certain coloring matters are altogether too slight. What an embarrassment, if the whole reticulum should be connective tissue, or horny material inert! Deiters' offshoots branch into it and axis-cylinders arise from it. Where are we to locate the centres of the sensitive nerves, if these inosculate with a connective-tissue reticulum?

There is but one way to escape all these difficulties, and this is to consider the reticulum as neither nervous nor connective tissue, but living or contractile matter. With this view an indirect connection is established between all central elements of the gray tissue, the nuclei, and the ganglionic corpuscles; though, I admit, the theory of a perfect insulation is lost. Similar formations we meet with in the retina, and there is no end of quarrels as to the nervous or connective-tissue nature of a number of filaments and reticular formations. Here, too, all difficulties could be overcome by simply admitting that the reticulum is living or contractile matter.

Quite recently an important confirmation of Gerlach's original assertion has been made by Béla Haller (1886).¹⁴ This author demonstrated, in the ganglia of molluscs, marginal ganglionic corpuscles and a reticulum emanating from them. Some of the offshoots of the latter directly become nerve-fibres, whereas the majority of the nerve-fibres originate from the reticulum. Thus we are positive of a double origin of nerve-fibres. L. Edinger¹⁵ is thoroughly convinced of this view, and we have good reasons to accept an excellent observer's coincidence with facts so much in harmony with the most advanced modern biological views.

More and more we approach the doctrine, first established by myself in 1873, which claims that the living matter is continuous throughout the whole animal organism. The

¹⁴ Untersuchungen über marine Rhipidoglossen: II. Textur des Centralnervensystems. *Morphol. Jahrb.*, XI.

¹⁵ Schmidt's *Jahrbücher*, Jahrgang 1887, No. 8.

apparently well-founded cell-theory must be sacrificed in order to obtain a plain understanding, not only of the action of nerve-tissue, but of the whole organism. The nerve-tissue is long since acknowledged to be continuous throughout the body: it will take but one step further to establish the continuity and life of all tissues constituting the animal and vegetable body.

In the United States the progress of the novel views, which are not the worse for being dubbed by the late L. Elsberg the "bioplasson-theory," is slow but steady. Charles F. Cox, in an excellent presidential address, delivered before the New York Microscopical Society, on January 3, 1890, expresses this progress in the following manner:

"I can well remember, as perhaps you also can, the disgusted incredulity with which this new doctrine was received—an incredulity in which, I confess, I then shared. I am not sure that the appearance of a reticulum in the prepared blood-corpuscle is even yet generally accepted as evidence of a normal structure of the kind claimed by Dr. Heitzmann; but the claim certainly gains support from the fact that vegetable histologists are pretty well agreed that a more or less similar reticulum is demonstrable in the protoplasm of plants. Prof. Goodale seems to have no doubt on this point. . . .

"In the work from which I have just quoted,¹⁶ Dr. Heitzmann generalizes as follows: 'What . . . was called a structureless, elementary organism, a "cell," I have demonstrated to consist only in part of living matter, while even the minutest granules of this matter are endowed with manifestations of life. The cell of the authors, therefore, is not an elementary, but a rather complicated, organism, of which small detached portions will exhibit amœboid motions. . . . How complicated the structure of a minute particle of living matter may be, we can hardly imagine; what we do know is that the so-called "cell" is composed of innumerable particles of living matter, every one of which is endowed with properties formerly attributed to the cell-organism.'

¹⁶ Microscopical Morphology. New York, 1883.

"It having been shown that life hangs upon a web of infinite tenuity, and does not reside necessarily in either a vesicle or a lump, it was a natural and easy step to extend this network from tissue to tissue and organ to organ, in an unbroken circuit of vital communication. This step Dr. Heitzmann does not hesitate to take; for, says he, 'there is no such thing as an isolated, individual cell in the tissues, as all cells prove to be joined throughout the organism, thus rendering the body *in toto* an individual. What was formerly thought to be a cell, is, in the present view, a node of a reticulum traversing the tissue. . . . The living matter of the tissues exists mainly in the reticular stage, and is inter-connected without interruption throughout the body.'

"Again, this at first very strange and, for some reason or another, unwelcome doctrine receives support from the investigations of botanists; for, as Prof. Goodale remarks, this protoplasmic inter-communication between adjoining cells 'has been shown to be so widely true in the case of the plants hitherto investigated, that the generalization has been ventured on that all the protoplasm throughout the plant is continuous.' The position to which we have traced this matter is, then, that to the latest biology, in any particular organism, a generally diffused and inter-connected substance, simple only in appearance under present optical aids, has taken place of the circumscribed, more or less isolated and independent, and recognizably complex vesicle which was the physical basis of life to the science of fifty years ago. In the words of Dr. Heitzmann: 'According to the former view, the body is composed of colonies of *amœbæ*; according to the latter, the body is composed of one complex *amœba*.'

Truth is welcome, from whatever quarters it may come. In the support of the recent views the botanists have proved to be superior to animal biologists. Still, the plants have no central nerve-organs, no nerves. How much plainer is the truth evinced by an unbiased study of the structure of the gray nerve-tissue!

HYSTERICAL FEVER.¹

BY MARY PUTNAM JACOBI, M. D.

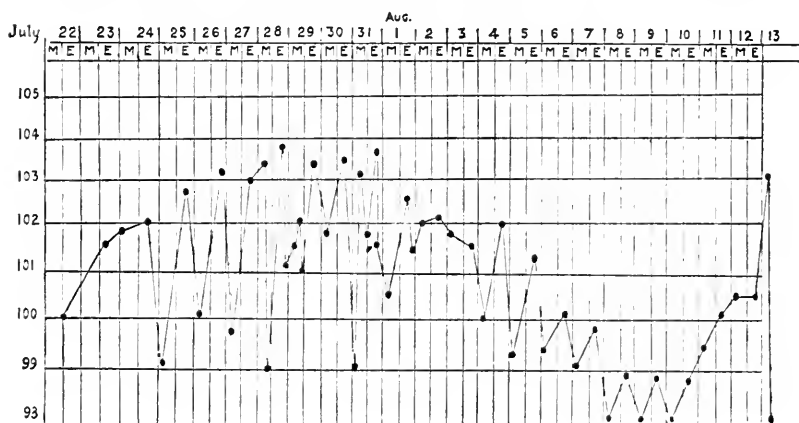
CASE M. M.—The illness to be described began on July 22d last, 1889, when the patient entered the New York Infirmary. But in the preceding year, winter of 1887–1888, she had suffered from a succession of disorders, to which reference must be made on account of their bearing on the illness in question.

In December, 1887, the patient was seized with a pain in the chest, unaccompanied by fever, but which was diagnosed pleurisy by the first physician consulted. His diagnosis had greatly alarmed the patient. As however, my own examination of the chest failed to discover any physical sign of pleurisy, I interpreted the pain as a pleurodynia. After this diagnosis, the pain rapidly subsided; but a paresis of the bladder, which had already showed itself, deepened to a complete paralysis and retention of urine. Catheterism was performed for some time, but the trouble finally yielded to strychnine and local faradization. There appeared severe pain in the left ovarian region, attended with fever. The temperature rose and fell irregularly through the day, occasionally going as high as 103°, more often reaching no higher maximum than 102°. Physical examination of the pelvic organs failed to discover any objective sign of local inflammation, and the disease finally subsided. Before the patient had left her room, however, she was attacked with a severe catarrhal sore throat, attended with abundant diffuse mucous exudation, but not truly diphtheritic. This was in March. After recovery and resumption of ordinary occupations, the patient became subject to intermenstrual metrorrhagia, for which no uterine cause could be ascertained, and which was referred to one of the obscure forms of functional ovarian irritation. During the summer of the

¹ Read at the Neurological Section of the Academy of Medicine, April, 1890.

same year (1888), the patient suffered from a bilateral partial paralysis of the lower extremities, especially affecting the peroneal muscles. She remained able to move her limbs in bed, but was quite unable to stand or walk. She recovered this power, however, when provided with braces which supported the ankles and reaching to the knees. She then went to the seashore, and for two or three months was perfectly well. On returning to the city and becoming involved in much mental worry and anxiety, her physical troubles returned. There were first, attacks of retention of urine and metrorrhagia; then almost entire inability to use the eyes in reading, which a competent oculist explained by simultaneous paresis of several external ocular muscles. He referred this, moreover to an attack of diphtheria which had been experienced five years before; but I think this was improbable, as, until the period which I have just described, the use of the eyes had been attended by no difficulty. It seems to me that the paresis was of a hysterical nature and analogous to that of the peroneal muscles, which had deprived the patient for a time of the power of walking. This opinion was subsequently also expressed by Dr. Putnam, of Boston. The ocular defect persisted through the winter. Twice during this time the patient was seized with an attack of severe pain in the abdomen, which, after lasting twenty-four hours, at once lost its acuity and rapidly subsided, when I had assured her with great positiveness that she did not have peritonitis. Once, after receipt of an agitating letter, she became apparently delirious and unable to speak for twelve hours. This was immediately followed by an intense dysphagia, overcome at last by a combination of moral force and local faradization. In the following summer, 1889, the patient's health was considerably improved. She engaged in some occupation involving considerable fatigue—I think teaching in a public night-school—and at once began to lose ground again. Early in July, during the second day of a menstrual period, she accompanied a friend on an excursion to Bedloe's Island, and climbed the stairs within the statue. The menstrual flow was immediately arrested,

and severe pain appeared in the left ovarian region. On the 22d of July she was admitted to the New York Infirmary, and on the 23d had a temperature of 102° , and the next day of 103° . The fever persisted at about this range till the 13th of August, when, after two days of normal temperature, it rose once more to 103° , and thence fell to a range between 98° and 101.5° , which it maintained till the middle of September. During this time I did not see the patient,



as I was absent from the city. The physicians in charge had only a very slight acquaintance with the previous history of the patient, and she herself gave an imperfect and rather misleading account of her series of illness. On account of the fever, the abdominal pain was explained by some focus of parametritis, but it was noted that the pelvic examination—made, it is true, with reserve on account of the acuity of the accidents—always failed to detect any evidence of inflammatory exudation.

On my return in the middle of September, the patient was in about the same condition as at the beginning of the attack, and quite the same as in the middle of August, after the fever had fallen to a low grade. Upon hearing the history and combining it with that of the many and varied attacks which I had previously and minutely observed, I ventured to express the positive opinion that on this occasion also no really inflammatory process had ever existed.

but that the accidents were nervous, and initiated by an ovarian irritation, the latter due to the arrest of menstruation by an unwonted physical exertion which involved the nerves of the lower extremities, *i. e.*, of the lumbar plexus, which also innervates, to a great extent, the ovary. It seemed probable that the menstrual arrest had left a congestion of the ovarian cortex, or even that minute hemorrhages had occurred there. The patient was put under ether, and a most thorough pelvic examination made, both by myself and by Dr. Cushier, with a completely negative result. Dr. Cushier admitted that the entire absence of any trace of exudation at this time, though some irregular low fever persisted and the abdominal pain was as severe as ever, rendered it altogether improbable that a parametritis had ever occurred.

With the concurrence of Drs. Cushier and Kilham, therefore, I positively assured the patient that she had no pelvic inflammation, that the attack was of the same nature as the others in which I had previously attended her; that she could safely get up from bed as soon as she pleased; and that a few applications of galvanism to the abdomen over the seat of the pain would rapidly dissipate it.

The applications were, in fact, made with the positive electrode over the ovarian region of the abdomen, the negative over the lumbar spine. Each application entirely removed the pain for many hours. But it seems probable that the moral effect of the diagnosis was quite as important, so rapidly did the patient change her attitude and so soon was she able to get out of the bed on which she had been lying for two months. In a week she was walking about; in ten days was entirely free from pain. The temperature remained normal from the day of the examination under ether.

Before the modern researches upon fever as the result of poisonous material circulating in the blood, the conception of a purely "nervous fever" was an entirely familiar one. Indeed the abdominal typhus, which is now recognized as a typical example of infectious disease, was considered, not so very long ago, as a "nervous fever," and

liable to be produced by causes which greatly fatigued or exhausted the nervous system.

The well-known urethral fever was an admitted case of a purely nervous fever of reflex origin. "Febrile movements" of all kinds were easily explained by varying functional irritations of the nervous system, among which were not reckoned irritaments conveyed to nerve centres in the blood nourishing them. Indeed, even the fever of inflammations was referred to the peripheric irritation of the nerves of the inflamed tissues; and not until much later was it suggested that some *materies morbi* was carried from the focus of inflammation to the central nervous system.

To-day, however, the point of view has so radically changed, that it is easy to forget that all the modern explanations of fever simply increase the list of irritaments to which the pyrogenic apparatus of the nervous system is susceptible. Although there be as there undoubtedly is, increased production of heat during fever, it is established that this would not cause a rise of body temperature unless the elimination of heat were simultaneously deranged absolutely or relatively. But this derangement in the elimination of heat depends upon disorder of the heat-regulating apparatus of medullary and cerebral centres, which thus react to the influence of the chemical poisons generated by inflammation or infection. There is, therefore, no essential contradiction between the new and old views about fever. An exclusively nervous cause is always plausible, because the proximate cause of increased body heat is always to be sought in the nervous system.

Before the thermometer was supposed to enable us to differentiate with precision between inflammatory and non-inflammatory pain, the liability of hysteria to simulate inflammations, and especially those of the abdominal cavity, was one of the well-worn themes of text-books. "Hysterical Peritonitis" is a classical chapter in every dissertation on hysteria, and in every guide to differential diagnosis in abdominal disease. But I think that to-day—and the case I have related shows it—we are liable some-

times to be misled by an habitual, though legitimate, reliance on the thermometer as a means of differentiation. It is easy to decide in the absence of fever that pelvic pain must depend upon some other cause than inflammation; and in the great majority of cases this conclusion is confirmed by the absence of all physical signs of exudation. Yet Dr. Thomas and some other gynecologists declare that an extensive pelvic exudation may be formed, and with considerable rapidity, without the slightest rise of temperature ever being produced. However this may be—and I confess never to have myself seen the statement proved—the two attacks of pseudo-parametritis attended by fever, which were sustained by the highly hysterical patient under discussion this evening, serve to illustrate the converse proposition, namely, that a rise of temperature may occur under circumstances strongly suggestive of pelvic inflammation and yet all positive proof of true inflammation be entirely lacking.

Hysterical fever has lately received much attention from both English and French physicians. In 1883 Pinard wrote a thesis on the pseudo-fever of hysterics, in which he claimed to show that hysterical fever did not really exist:—that is, in the cases described: either no thermometrical observation had been taken, or the thermometer registered a temperature not above 38° C., while often the temperature remained normal. The pseudo-fever consisted, therefore, in an assemblage of symptoms which simulated fever, but were not truly febrile. Among these was conspicuous the acceleration of the pulse, phenomenon essentially analogous to the tachycardia of exophthalmic goitre. The patients often had subjective sensations of heat, also severe headache and coated tongue. This condition was not unfrequently regularly paroxysmal, so as to simulate attacks of malarial fever, but was entirely uncontrolled by quinine.

In a more recent thesis, passed by Henri Fabre in 1888, the existence of a true fever, and even hyperpyrexia of really hysterical origin, is, however, formally reasserted. Cases are related where such fever was accompanied by functional disturbance of various organs, so as to simulate

respectively meningitis, peritonitis, or pneumonia. Intermittent fever and typhoid fever are also said to be simulated. The same assertion is made by an American physician, Bressler, in a communication to the "Medical Record," for 1888. This writer relates no cases in detail, and I do not think that his diagnosis is absolutely proved by his descriptions.

"By hysterical fever," says Dr. Bressler, "I mean a perverted condition of the nervous system, occurring in a neurotic individual, attended by an elevated temperature, which may last from a few hours to several days, and is associated throughout its duration with symptoms of an hysterical character." "This fever," continues the writer, "generally begins with symptoms simulating a mild intermittent—chilliness, loss of appetite, constipation, or occasional diarrhœa; tongue coated, headache, general malaise, rise of temperature, face flushed generally, or in a circumscribed spot on the cheeks, eyes clear and brilliant, mind bright, comprehension quickened. There is general muscular and cutaneous hyperæsthesia. The special senses are more acute; there is no true delirium. The stomach is excessively irritable, and vomiting very persistent. The abdomen is extremely sensitive to pressure, and peritonitis may be simulated, but may be excluded by the fluctuating character of the pains, the absence of tympanitis, and the development of ovarian pain under pressure. The temperature varies from 101° to 105° F., and the maximum is reached early in the attack."

In the "Transactions of the London Clinical Society," Dr Hale White relates the following case: A girl of eighteen was admitted to the ward, on the 10th of August, for a febrile attack, which lasted four days, and then subsided. On September 8th she was suddenly taken ill with a severe pain in the left side, and was readmitted to the hospital the next day. The patient could hardly walk, and was somewhat incoherent in speech. Within the course of twelve hours the pain was located in four different places—the left iliac region, the epigastrium, the lumbar region, the splenic region. The attention of the patient was easily diverted by

conversation, and she then permitted considerable pressure over the seat of the pain. The temperature was at first 103° ; on September 10th, after a chill, rose to 105° , to fall in the evening to 99° . On the 11th, at 6 A. M., the temperature was 98.6° , at 6 P. M. 104° ; September 12th the temperature did not rise till evening, when it was 102° at 6 and 98.8° at 10.

In the analysis of the case Dr. White excluded all other causes of either the pain or the fever except hysteria. But it is noticeable that the patient vomited on two successive days, and during the previous brief illness in August there had also been symptoms of a gastro-duodenal catarrh. It seems to me that such an organic condition really existed, and was the immediate cause of the neurotic condition upon which the wandering pains, and markedly irregular fever, directly depended.

Dr. White remarks that, although several cases of hysterical pyrexia have lately been recorded, much skepticism has been expressed in regard to it. Among these recorded cases is one by Clemrow, in the "Medical Press and Circular," of 1887. A laundrymaid, of twenty-three, was admitted to the Edinburgh Royal Infirmary, October 22d, with dizziness, pain in the left side, and a purpuric rash over the lower extremities. On the 29th of November the patient had a severe fright, and her temperature rose to 107.8° . After this the records of temperature are so extraordinary as to suggest fraud, were it not that there was no way in which a fraud could have been effective. At midnight of the same day three successive records, taken at short intervals, read 111° , 108° , 98° . On November 30th the temperature in the right axilla was 108° ; the left, at the same time, 99.4° . At midnight the temperature was 98° on the right side and 108° on the left. Similar local maxima, varying from hour to hour, were observed on the 1st, 2d, and 3d of December; after which the records are not given. On November 30th the patient had several spasms simulating tetanus, probably hysterical opisthotonos. On December 1st, together with headache and nausea, there was a peculiar rhythmical movement of the eyelids, alternate

elevation and depression. There was left internal strabismus, and sluggish reaction to the light of the right pupil. Throbbing pain at the vertex increased by pressure. On December 3d there were frequent spasms, with muffled heart-sounds; pulse at the wrist imperceptible. On December 4th the patient became delirious, and continued so until the 13th. The plantar and patellar reflexes were both absent; there was cutaneous anæsthesia, incontinence of urine and fæces. After the 13th these symptoms disappeared, and the patient began slowly to improve. But she was not fully recovered until April.

Clemrow considered the hyperpyrexias to have been local, and not extending throughout the body.

In the "*Lancet*," for 1879, Donkin related the case of a girl of nineteen, who, during convalescence from a mild typhoid fever, had, at frequent intervals, temperatures of 108° or 110° . These were of short duration, and unaccompanied by other symptoms than a sensation of heat.

In another case, observed by the same writer, from the 20th of May to the 20th of June the temperature every morning and evening varied between 101.8° and 106.8° .

Donkin quotes similar cases from Creig Smith, Cliffe, and Meade. The last, like Donkin's own case, was also a girl convalescent from typhoid, whose temperature for a month kept incessantly varying from 103° to 109° , sometimes in fifteen minutes would run up to 111° . In these English cases the temperature was always taken in the axilla.

In the "*Gazette Hebdomadaire*," for 1886, Debove describes a patient who, every day for a month, and without other symptom, presented morning and evening a temperature of 39.5° C. This was in November. In December the temperature rose to 40° , on the 17th of January was 41.4° and on the 24th reached a final maximum of 41.4° . After this it slowly fell, and became normal on the 30th. During this period of three months the morning and evening temperatures were almost always alike: occasionally one or the other was higher by one-tenth or two-tenths of a degree. This prolonged hyperpyrexia resulted in no emaciation or loss of strength.

In 1886 Barié described a case (also in "*Gazette Hebdomadaire*"), a severely hysterical young woman, servant at Bicêtre. She was subject to frequent convulsive attacks, transient paralyses, profound disorders of sensibility. One morning, after a violent convulsion, she became completely hemiplegic, on the left side, except the face, as regarded both mobility and sensibility. After this she had thirty convulsive attacks in the course of twelve days. Sometimes for two or three days together she would remain in a state of complete mutism, without eating and also without urinating. All remedial measures failed, and the physician contented himself with simple observation. One morning, after a violent convulsive attack, the temperature in the axilla was found to be 39° C. From this time, for twenty days, there was permanent fever, as measured both in the axilla and rectum. Evening temperature was usually higher than morning by some tenths of a degree, but on five days the morning temperature was the highest. There was no functional disturbance, and the tongue remained moist. The fever was highest on the days of the attacks, but persisted on the other days also. On the twentieth day sudden defervescence occurred, the patient remaining otherwise the same, neither better nor worse.

In the "*Periscope*" of the *JOURNAL OF NERVOUS AND MENTAL DISEASE*, for February, 1890, is described a case of hysterical pseudo-phthisis where, during three days, the temperature varied from 103° to 104° F.; on the fourth day it rose to 113° , and the patient became slightly delirious. In an hour the temperature fell to 108° ; in the evening was 106.3° . On the next day it again rose to 113° , but fell in an hour to 99.5° . During the next few days the temperature varied from 101.3° to 103.1° , and then became normal. The symptoms had begun with an attack of hæmoptysis, which was followed by severe dyspnœa, cyanosis, and apparently threatened asphyxia several times during the night. During the next two months the same group of symptoms was repeated several times with complete absence of physical signs of phthisis. There was retention of urine.

The most interesting cases quoted in the thesis of Henri

Fabre are two, of simulated meningitis, one of apparently severe pulmonary disease. The first of these, a young woman of twenty-four, who had previously suffered from chorea and nervous aphonia, was admitted to the hospital with a temperature of 39.5° C. Her face was swollen and congested, eyes closed on account of an intense photophobia. The head was retracted completely, cephalalgia violent, insomnia and cries, abdomen retracted, constipation absolute, meningitic streak easily developed, severe generalized hyperæsthesia, knee-jerk little modified, no morbid condition discoverable in lungs, heart, or kidneys. During ten days the patient remained in about the same condition: prostrated, eyebrows contracted, pupils contracted but equal, five or six times bilious vomiting without effort (having all the appearance of cerebral vomiting). A diagnosis was made of tubercular meningitis, and (but with little hope of doing any good) leeches were applied behind the ears and calomel administered. On the tenth day the patient was found sleeping naturally, and, on being aroused, ceased to complain of the pain in her head. The temperature had fallen to 38° C. In a few days more the patient was fully convalescent, but on first getting up was affected by a transient paraplegia.

The history of the second case closely resembled the first.

I have myself seen a similar case in the service of Cornil at La Charité, and, curiously enough, the same patient returned, a year later, with the same group of symptoms, and, her personality being recognized, the diagnosis was the second time at once correctly made.

The case of febrile hysteric dyspnœa related by Fabre is as follows: The patient was a woman of twenty-six; admitted to the hospital with an evening temperature of 39° C. and a dyspnœa of five or six days' duration. There were thirty-five to forty respirations a minute, but unaccompanied by trace of cyanosis. The most careful auscultation failed to discover any lesion of either lungs or heart, and the absence of albuminuria was held to exclude a uræmic origin to the dyspnœa. The fever continued for twenty

days, being extremely irregular, with occasional intermissions of normal temperature, followed by a rise to 39° or 40° or over. On the twentieth day occurred an abrupt deferescence, and at the same time the dyspnœa ceased.

The recognition of hysterical fever as a distinct clinical affection has been much facilitated by recent researches on the relations of the cerebro-spinal nerve-centres to the temperatures (general or local) of the body. As every one knows, these researches were initiated by the famous observation of Sir Benjamin Brodie, on a rise of temperature in a few hours to 111° F., in a patient who had sustained a fracture of the spine, with traumatic section of the cord. This observation was published in the "Medico-Chirurgical Transactions" in 1837.

The researches of Tscheschin, in 1866, are equally famous and well known. In some respects they seem in contradiction with Brodie's clinical observation: for when, in animals, this experimenter cut the spinal cord below the medulla, the temperature of the body fell; but if the section were made between the medulla and the pons, the temperature rose excessively.

The more exact experiments of Horatio Wood, in his beautiful researches on fever, published in 1880, demonstrated that when the spinal cord was cut anywhere between the level of the third and second cervical vertebra there was at first an enormous increase of heat-dissipation, correlative with the general vaso-motor paralysis; that in forty-eight hours this was followed by a diminution in the dissipation of heat, but also a diminution in heat-production, so that, as had been before observed, the net result was a fall of body temperature. Wood also observed the rise of temperature consecutive to section of the cord between the medulla and pons. He accepts the inference drawn from the facts by Tscheschin, that there exists in the medulla some nerve centre or centres whose influence tends to stimulate the production of heat in the thermo-genetic tissues, namely, the muscles; that this influence is habitually restrained by that of moderating centres in the pons or above it, and that the rise of temperature observed in

the last experiment is due to the withdrawal of this moderating influence from the real heat centres. More recent experiments have extended the field of experiment and inquiry. Eulenburg and Landois showed that excitation of one cerebral hemisphere is followed by a local rise of temperature in the limbs of the opposite side. These experimenters made no observations on the general temperature. In 1884, Charles Richet (*Compt. Rend. Societé Biol.*, 22 Mars, 1884) pricked one cerebral hemisphere of a rabbit with a steel pen which perforated the cranium, and found in the course of two hours that the rectal temperature rose from 39.5°C , to 40.4° . The next day, when the temperature had fallen to 39.2° , a nerve pricking caused a rise to 42.8° . The animal died in the night, presumably of the hyperpyrexia, as no brain lesions were discovered to explain the death. It was found that the pin had penetrated to a spot situated three or four millimetres in front of the corpus striatum.

A little later, Schreiber² found that a rise of temperature occurred after lesion of any part of the pons, of the cerebral peduncles, cerebrum or cerebellum, provided the animal operated on were protected from the radiation of heat by wrapping in cotton wool. In 1885, Aronsohn and Sachs in Germany, and Dr. Isaac Ott in America, began almost simultaneously, but quite independently of each other, to search for heat-regulating centres in the brain. The German observers³ trepanned rabbits at the juncture of the sagittal and coronal sutures, and entered the brain with a needle, three millimetres broad, at a point about one millimetre outside the longitudinal sinus. A carbolized dressing was immediately applied, and the well-being of the animals seemed to remain undisturbed.

When the operation was performed on the cerebrum anterior to the Rolandic convolutions, no effect on the temperature was observed. But the punctures which passed to the base of the brain, from the point of junction of the coronal and sagittal sutures, were always followed by an

² Pfluger's *Archiv.*, viii., S. 576.

³ Pfluger's *Archiv.*, 1885.

enormous rise of temperature. If the puncture only penetrated the cortex cerebri, no effect on temperature was produced. Electrical irritation of the susceptible region, *i. e.*, the tissue just in front or on the outer side of the corpus striatum, also caused a rise of temperature. An increased excretion of nitrogen was observed during this artificial fever, so an increased heat-production was inferred, but no calometrical observations were made.

These difficult observations were, however, made by Ott,⁴ and add greatly to the value of his experiments on the brain.

Ott established four localities at the base of the brain whose puncture, and consequent irritation, was followed by a rise of body temperature. These were, at a point just within the anterior part of the corpus striatum; a second point between the corpus striatum and the thalamus; a third at the anterior part of the thalamus; and a fourth at the point of decussation of motor fibres at the nib of the calamus in the medulla. In the fever consecutive to irritation of these centres, there is at first an increase of both heat-production and heat-dissipation, but both soon fall below normal, though fever continues. In addition to these centres, however, Ott discovered two others on the cortex; one at the point of juncture of the supra sylvian and post sylvian fissure; the other in the neighborhood of the cruciate sulcus, *i. e.*, over the Rolandic convolutions.

When either of these cortical centres were irritated, temperature was depressed. If, on the other hand, they were removed by slicing and subsequent washing with carbolized water, the temperature rose.

From the total result of his experiments, Ott infers that the basal centres, like those of the spinal cord, habitually stimulate the production of heat; are thermogenetic centres. But those of the cortex, the sylvian and cruciate, habitually restrain the activity of these lower centres, and may therefore be called thermotaxic.

Under certain circumstances the striate and extra striate centres may also be thermotaxic, and moderate the

⁴ Journal Mental Disease, 1888.

spinal centres below them. They have, therefore, a mixed character or function.

Girard⁵ confirmed the results of Ott's experiments on the corpus striatum, and also observed a rise of temperature to follow punctures at various localities in the posterior part of the brain, but none when these were made anteriorly. The fever was attended by increased elimination of nitrogen in the urine, and was controlled by antipyrine. Rise of temperature was also induced by faradising the striated bodies for half an hour with needles insulated to their tips.

Horatio Wood, also, in thirteen out of fourteen experiments, found that localized destructions of tissue just behind the crucial sulcus, thus compromising Hitzig's region, were followed by a rise of temperature and decided increased of heat-production.

A curious confirmation of the foregoing observations is offered by Zawadowski,⁶ who found that antipyrine ceases to reduce temperature if administered after section of the spinal cord at the atlas, an operation which removes the inhibitory influence of the brain from the thermogenetic centres of the cord.

The interest of the foregoing observations is very great in their bearing on the general theory of fever. In accordance with them, all fever can finally be ascribed to derangement of the central nervous apparatus, which controls the generation of heat in the muscles, the latter being the ultimate thermogenetic apparatus. Hence, the striking fact, that the cerebral centres so far established as regulating the production of heat, are chiefly situated on the motor tracts, namely the Rolandic convolutions, the striate centres, and the medulla.

In zymotic fever the thermogenetic centres would be irritated by the poison circulating in the blood; in traumatic, perhaps also in inflammatory fever the same result is produced by irritation of peripheric nerves; in hysteria there would be paralysis of the cortical thermotaxic inhibitory centres rather than excitation of the basal thermo-

⁵ Archives to Physiol., 1886 and 1888.

⁶ Centralblatt f. medicin-wissen, 1888.

genetic centres.⁷ Reflex fevers, like urethral and worm fever, might be supposed to imply, on the other hand direct irritation of the thermogenetic centres.

This paralysis would then enter into the entire series of hysterical phenomena, which depend upon loss of cortical control over lower centres. It becomes analogous to the loss of cortical control over subcortical vaso-motor centres, upon which Meynert has so strongly insisted, and nevertheless it is not to be resolved into a vaso-motor phenomenon. For it has been shown, especially in some experiments of Wood's, that the vaso-motor medullary centres are not affected in these artificial fevers, and respond as usual to an irritation of the sciatic nerve.

A danger attends the recognition of any group of clinical symptoms as hysterical. It is the danger of ascribing to hysteria, symptoms which are really caused by organic disease. This is even more serious than the opposite error of interpreting as the result of organic disease, symptoms really due to hysteria. The diagnosis is, therefore, always important, and often delicate and difficult. It would be impracticable in this place to analyze the elements of diagnosis in regard to each case which might be simulated. But this may always be remembered: Exclusion of the grave organic lesion which may be simulated, does not necessarily exclude the origin of the disorder in some lesser lesion, which may even entirely disappear, while the storm which has been aroused continues. The type of such a sequence is offered by the prolonged hysterical neuralgias which may originate in a slight sprain (traumatic hysteria).

In the case which forms the basis of this paper, I think it is not at all improbable that the last series of accidents originated in a slight hemorrhage into the cortex of the ovary, occurring at the time of the arrested menstruation. A permanent ovarian irritation or irritability existed, manifested by the persistent recurrence of menorrhagias, in the absence of all uterine disease. It seems as if this would be sufficient to explain the entire series of phenomena, itself being an expression of a grave hysterical diathesis.

⁷ W. Hall White, loc. cit.

A CASE OF CHOREA ATTENDED WITH MULTIPLE NEURITIS.¹

By FRANK R. FRY, A.M., M.D., of ST. LOUIS.

BARBARA MUELLER first presented herself at the clinic for diseases of the nervous system, at the St. Louis Medical College, January 23, 1886. She was then eleven years of age. Nothing of importance in family history was obtained, except that her father, who always accompanied her to the dispensary, occasionally had attacks of sub-acute rheumatism; and that her younger brother had been treated by us for chorea, as I shall hereafter explain.

She had a general chorea, attended by no unusual features that were then discovered. The movements were more pronounced on the right side of the body, including the face. A slight paresis was apparent in the extremities of this side when the choreic movements were disappearing. This was transient. When she first came she was anæmic, restless, sleepless, and very irritable. These symptoms soon began to disappear. She made a rapid recovery, and was discharged within a few weeks apparently well, having gained considerably in weight and greatly improved in general appearance. The treatment consisted of arsenic and iron, and, at first, bromides at night.

March 24, 1887, she returned to the clinic. Her father stated that she had appeared to be very well until a few days prior to this date, when she complained of starting from her sleep at night. Her appetite was failing; she was becoming very restless and peevish, and complained of the lumps on her legs. There were decided but feeble general choreic movements of the whole body. On the lower extremities

¹ Read before the Medical Association of the State of Missouri, May 7, 1890

there was a typical erythema nodosum, of which she complained considerably. The heart's action was feeble and rapid. There was no murmur. She did not seem to be so anæmic as when she came the year before, but she showed evidence of a general debility or lassitude which contrasted strongly with her condition when she had discontinued treatment the previous spring.

April 9th, after a careful examination, I found that none of the tendon- or skin-reflexes in the extremities could be produced. I did not try the nose or pharynx. I noticed for the first time on this date an inequality of the pupils, the left remaining decidedly larger. Dr. William Hunicke made an examination, and was unable to assign a satisfactory cause for the inequality, but thought it possibly due to some central disturbance. The choreic movements became a little worse, but were not severe enough to cause much inconvenience. All of the symptoms, including the chorea, soon began rapidly to disappear, and she discontinued her attendance at the dispensary in the latter part of May (1887), apparently well.

January 3, 1888, she returned again to the clinic. She had a return of the chorea, which was general but not severe. Her general condition seemed better than when she applied for treatment on the former occasions. Her visits were not frequent, and nothing remarkable was noted until

February 4th, when she came with a marked paresis of the lower extremities, which, so far as could be learned from the father, had developed within a day or so. The dropping of the toes in walking, or the gait characteristic of paralysis of the anterior tibial nerves, was remarked. The knee-jerk was gone. She complained of tingling in the feet. Arsenic was discontinued.

February 10th she was unable to walk, and was carried to the clinic. She complained of painful tingling in the feet and legs and to some extent in the hands. Muscular power in the hands and forearms was very feeble. From this date her visits to the clinic were infrequent.

April 12th (two months after the declaration of the

paralysis) the first careful electrical examination was made. It showed a reaction of degeneration in the muscles of the hands, feet, forearms, legs, arms, and thighs. The muscles were considerably atrophied and already somewhat contracted, the toes being flexed and the ankles extended, the hands and wrists presenting the first stage of the well-described bird-claw deformity. Tactile and temperature sense were almost *nil* over the areas of motor paralysis. The deep and superficial reflexes were gone. The muscles of the face, neck, and trunk were not involved; neither was sensation disturbed over these areas.

May 1st there was the first evidence of improvement, she being able to slightly move the arms. From this time improvement was continuous, but very gradual.

May, 1889 (fifteen months after commencement of paralysis), she was walking very well, the toes, however, dropping as she raised the feet in stepping, giving an appreciable halt to the gait, although she could walk fast. The deformity was fast disappearing from the hands. The muscular power in all the extremities was good.

I last saw her in November (1889). There was then a barely appreciable dropping of the toes, suggesting more of a stiff gait from tight shoes than a paralysis or paresis of the anterior tibial nerves. The usefulness and shape of the hands were completely restored. So far as I could determine, sensation was about normal. The knee-jerk was still absent. Her general condition was very good.

The immediate cause of this paralysis was undoubtedly a multiple neuritis. That it was not a cerebral paralysis is apparent; for, besides its character—*i. e.*, an atrophic paralysis with the reflexes gone—it had not the distribution of a cerebral paralysis. That it was due to disease of the peripheral portions of the spinal nerves, and not of the cord, an examination of the facts makes clear: It was symmetrical, so much so indeed that the deformity of the hands was almost identical on the two sides, and also the amount of flexion of the toes. It was not only symmetrical, but the paralysis was most profound in the distal portions of the several extremities, lessening toward the trunk. The sen-

sory disturbance was extensive—in fact, coextensive with the motor paralysis, and persistent. Although the paralysis was profound and extensive and accompanied with atrophy, the recovery was complete.

The question now comes, what was the cause of the neuritis? It could not have been of alcoholic origin, as there is positive evidence that none was taken. There had been no exposure to cold or wet, to account for it. A neuritis of this description is said to sometimes accompany or follow rheumatism; but our patient had never had rheumatism nor any articular trouble. It was suggested at the time that it was possibly due to arsenic, which she had been taking for a month, prior to which time she had had none for eight months. She had taken larger quantities for a longer time on former occasions, had borne it well, and improved during its administration. The drug was entirely and permanently discontinued on the appearance of the paralysis; yet the latter ran a tedious course, as described above. That the neuritis simply followed an exhausted or depreciated condition of the system, caused by chorea, as it sometimes does typhoid, phthisis, etc., may not, I think, be too readily conceded. In the first place, the association of multiple neuritis with phthisis, typhoid, diphtheria, and similarly debilitating diseases is by no means clear. In the second place, there were no unusual evidences of exhaustion or vitiated condition of the system on this occasion; in fact, when she was seized with the paralysis, her general condition seemed better, as stated in the history, than on the occasions of her former attacks of chorea.

Before attempting to look further for a possible etiological factor, I shall briefly relate, as a matter of incidental interest, our experience with this girl's brother. Fourteen months prior to her first visit to us he was brought to the dispensary with his first attack of chorea. He was then seven years old. He has returned once or twice every year since to be treated for the same trouble. On most of these occasions there has been an unusually rapid and marked change in his general condition following the administration of arsenic; beyond this nothing especially remarkable until

last fall. At this time there were, for the first, pains in the joints, and finally sub-acute arthritis with swelling and slight fever. Later still a soft murmur appeared in the heart, and persisted for about three weeks. The pulse was rapid, irregular, and weak. These symptoms all disappeared, and when he left us he was as well as I have ever seen him.

Cases of chorea occasionally occur which suggest, in some respects at least, the possibility of an infectious origin. Some observers have been so impressed with the fact, that they have undertaken laborious investigations looking toward the discovery of unknown factors, possibly infectious, in the etiology of this disease. In a paper² which I read before the Mississippi Valley Medical Society four years ago, I reviewed at some length a paper on the "Prechoreic Stage of Chorea," read by Dr. C. R. Stratton at the annual meeting of the British Medical Association in 1885. He reminds clinicians of the fact that a considerable proportion of the young subjects in whom chorea appears are found to have been in a prodromal stage, so to speak, characterized by anæmia, general lowered vitality (accompanied often with sores on the margins of the lips and nose), blunted intellect, great physical and mental irritability, sometimes by slight febrile action, vague pains and swellings of the joints, heart-murmurs, etc.; and that, in this condition, they are often treated for malaria, rheumatism, general debility, etc., until the chorea appears. With these clinical facts in mind, he suggests that chorea may be not a constant but an occasional result or symptom of some malady or maladies, possibly infectious, whose characters are not yet known to us. He made examinations of the micro-organisms found in the sores on the lips and nose, and, in post-mortem cases, of the vegetations found on the valves of the heart, and of certain minute infarctions found in the nerve-centres. In these vegetations he found the same organisms that he found in the sores on the face; and he believed the infarctions to be formed from small particles carried from the

² St. Louis Courier of Medicine, August, 1886.

valve-lesions to the distant capillaries, forming in the brain-tissue, and probably elsewhere, a characteristic pathological condition. Although the microscopical findings proved nothing definitely, Dr. Stratton was of the opinion that they lent color to his suggestion. In other words, the presence of the same micrococcus, which he then believed could be proved to possess distinctive characters of staining, etc., in these several regions of the body, would seem to supplement very well the clinical features sustaining his theory of an infectious origin.

This girl and her brother repeatedly appeared at the clinic, as the records show, in this same anæmic condition, with restlessness, sleeplessness, and mental excitability, with disturbed heart-action, with an unusual eruption in one instance in the girl's case and an arthritis and heart-murmur in the boy's case. In both these cases sores on the margins of the lips and nose were observed and noted, but were not invariably present during the choreic attacks. The great and seemingly unaccountable change in the condition of these two patients after a few days or weeks of treatment was quite remarkable; in the boy's case especially on two occasions amounting almost to a transformation, and a rapid one at that. I do not refer to the disappearance of the chorea merely, but to the improvement in general condition. Like other observers, I have seen, during a clinical experience of ten years in the city, very rapid improvement in choreic cases. But I have never seen it so prompt and striking and equally unaccountable as in one at least of these two cases, which have been under my observation, one five, the other four years. If I were looking for cases to classify in a category of cases of possible infectious origin, I should select these. I have seen others which less forcibly impressed me in the same way. Was the extensive multiple neuritis in our case possibly due to an infectious cause?

NOTES ON THE ACTION OF GELSEMIUM IN SOME LOCAL SPASMS AND NEURALGIAS.

BY M. IMOGENE BASSETTE, M.D.

Instructor in Electro-Therapeutics and Clinical Assistant in the Department of Diseases of the Nervous System in the Philadelphia Polyclinic; Clinician to the Gynecological Department of the Woman's Hospital of Philadelphia.

DURING the last two years I have had opportunities at the Polyclinic, and to a less extent at the Woman's Hospital and in private practice, of observing the effects of gelsemium. From this experience the drug seems to be serviceable in the treatment of some local spasms, some neuralgias, and a few distressing and unpleasant head symptoms, such as a feeling of fullness, beating and throbbing. That the efficient dose of the remedy, and by this I mean of the same preparation, varies through wide limits for different individuals is one of the most striking facts in reference to it.

The first and chief case to which I will refer in these notes was one of spasmodic torticollis, a patient who came to the Polyclinic in November, 1889, having developed clonic spasm in the muscles supplied by the spinal accessory nerve after an accident in which she was thrown out of a wagon. She was used by Dr. Mills as a text for a lecture at the Philadelphia Hospital, a report of which was published in the "University Magazine," February, 1890, and the details of the case can be found in this article. The spasm was very severe in character, and was brought on and increased by walking, excitement, or by pressure in the left occipital region. It stubbornly resisted treatment by such means as potassium iodide and bromides, mercury, salicylates, arsenious acid, assafœtida, sumbul, blistering, and the actual cautery.

The present notes refer almost entirely to the use of gelsemium, which was carried to an extreme and with

great benefit. She was first placed upon the fluid extract of gelsemium in doses of five drops four times daily; and this dose was increased daily by one drop until she took twenty-four drops four times daily, up to which time no constitutional effects were visible. She was then placed upon Parke, Davis & Co.'s normal liquid gelsemium, beginning with five drops three times daily. The dose of this was also increased until she took twenty drops, when she began to see double and feel dizzy. The drug was omitted for one day, and she was then again put upon twenty drops, and this was increased to twenty-five with no effect. (Two drops of fluid extract of gelsemium are equal to about one minim.)

At this time, January 18, 1889, she went to the Woman's Hospital, under the care of Dr. John B. Roberts, who placed her upon Shoemaker's fluid extract of gelsemium, beginning with five-minim doses. January 19th the dose was increased to ten minims; this was further increased between the 19th and 31st, until she was taking twenty minims—she then complaining of dizziness and disturbance of vision, once seeing double, but these symptoms lasting only a short time. On the 31st she began to take seven minims every two hours, day and night, this to be increased one drop every other dose after the first twelve hours. The next day, February 1st, decided constitutional effects were obtained, the patient remaining all day in bed with double vision and considerable headache. These symptoms passed away and gelsemium was continued and increased, and by the 4th she had reached fifteen minims. The dose was then reduced to thirteen minims, and kept at this every two hours both day and night. On the 8th the constitutional effects of the drug were again marked, and there was now some improvement in the spasm and position of the head. She continued until the 21st taking thirteen minims every two hours and steadily improving. She was now able to walk out without suffering, the spasm being better, the head much straighter and the muscles softer.

On the 21st the frequency of giving the gelsemium was reduced; the thirteen minims being now given every two hours during the day—the first dose at 7 A. M. and the last at 7 P. M.—and only twice during the night, at 11 P. M. and 3 A. M. This treatment was continued forty-seven days until she left the hospital. Massage and sitz-baths were also used, and March 4th, tincture of chloride of iron, fourteen drops an hour after each meal, was ordered. Heat and pressure to the affected muscles were also used. April 9th the patient was discharged, still taking the same dose of gelsemium and tincture of chloride of iron. I have heard from her within a week, and she still continues better, but has not taken any gelsemium for nearly three weeks.

For three weeks, from January 31st to February 21st, this patient took for the greater part of the time twenty-six drops or thirteen minims every two hours night and day. For another part of this time, alternately, either thirteen minims, or seven and a half minims every two hours, or about this amount. For the remaining forty-seven days she took about thirteen minims eight times daily. The average taken daily, when the maximum amount was administered, was about one hundred and fifty-six minims. During three weeks, from January 31st to February 21st, she probably took between five and six ounces, and during the last forty-seven days in all 4,888 minims, equal to eighty-one ounces.

A case of this kind may be worth reporting, both on account of the great benefit derived from the use of gelsemium and as a study of the dosage of this drug. It is well known that local clonic spasm is usually very obstinate. I have seen two severe cases of spasmodic torticollis besides this one. In one of these an inch or more of the accessory nerve was resected, but the spasm returned with full force; in the other, myotomy was performed, but with no benefit. The latter case made remarkable improvement under large doses of gelsemium. Numerous cases have been operated upon, but without success. The actual cautery, although useful, often fails; it was thoroughly tried in the case just reported. Such facts afford an excuse for heroic drugging.

A woman, B. R., thirty-four years old, brought to the Polyclinic by Dr. Bunting, was an illustration of one of the forms of painless tic, or spasm of the muscles supplied by the seventh nerve. Three years before she noticed a twitching under the right eye; this gradually spread over the lower face. Most of the time the lower eyelid was twitching spasmodically, and at the same time the mouth was drawn upward and outward. When talking or under any excitement, the spasm increased, so that the eye closed entirely, and the face was strongly contracted. The muscles chiefly affected were the zygomatics, the levators of the angle of the mouth, and the lower half of the orbicularis palpebrarum. When the attacks were severe she complained of pain and discomfort in the face. She was put upon two minims of the fluid extract of gelsemium, four times daily, with directions to increase the dose until constitutional symptoms were obtained. When eight minims were reached she began to complain of dimness of sight, dizziness, and a feeling as if she were drunk; but the spasm was much better than it had been for months, and the local sense of pain and discomfort had disappeared. She was now ordered to begin again with five minims four times daily and increase gradually. When seven minims were taken she became dizzy, her sight was dim, and her limbs felt almost helpless; but the spasm had almost disappeared. The dose was reduced to three minims; and later increased to four minims without physiological effects, when the spasm again getting worse, the dose was pushed up to seven minims, but the effects were so distressing that the gelsemium had to be stopped for a week. She is still under treatment.

In some cases of local spasm patients seem to stand large and long-continued doses of gelsemium, much as those suffering from severe pain will endure enormous doses of opiates or other narcotics. A gelsemium habit is not formed, although the drug is taken in immense doses and for a long time, one and perhaps the principal reason for this being its unpleasant effects.

In certain neuralgias, particularly those of the upper branches of the fifth nerve, both the therapeutic and toxic

effects seem to be obtained with much smaller doses. In three such cases decided relief of pain was produced quickly by the administration of a comparatively small dose of the fluid extract of gelsemium. It would be a great gain if we could separate those cases of neuralgia in which the drug has a favorable action from those in which it proves inert or of but little benefit. In one case the patient complained of great facial pain, stiffness of the jaw, and pain in opening and closing the mouth. She was put upon three minims of the fluid extract, the dose to be increased, and by the time she had reached six minims the pain had disappeared, and the relief was probably permanent, as after several weeks she has had no return of the symptoms. In two other cases of ciliary and supra-orbital neuralgia the pain was relieved by doses of three minims.

Gelsemium is a drug about which differing reports and differences of opinion are common. Cases of poisoning are reported which show the small amount of this drug which will produce toxic or fatal effects. Boutelle¹ reports a case of fatal poisoning in a man, aged twenty-four, who took for neuralgia a teaspoonful of Tilden's fluid extract of gelsemium and repeated the dose in fifteen minutes. Friedrich² reports the case of a girl of fourteen, who took a teaspoonful, and in less than an hour began to show physiological effects, later convulsions, and still later unconsciousness came on; but under active treatment she recovered. Sinkler³ reports the case of a woman who had serious toxic symptoms from taking five drops three times daily for ten days, the first dose having produced physiological effects. Dr. De Wolfe,⁴ for facial neuralgia, took ten minims of the fluid extract, and repeated this in half an hour. In less than fifteen minutes he was drowsy and could not keep awake; he was taken with shivering, dizziness, and symptoms of collapse; but under the use of stimulants the unpleasant symptoms passed away.

¹ Boston Med. and Surg. Journal, 1874.

² Philadelphia Medical Times, December 30, 1882.

³ Philadelphia Medical Times, January 5, 1878.

⁴ British Med. Journal, vol. i., 181, p. 193.

In the "Therapeutic Gazette," for June and November, 1889, and January, 1890, are some notes and queries, about the use of gelsemium, which bring out a few interesting points with reference to its dosage. Dr. Hutchings gives some personal experience of the drug, and considers a dose of fifteen or twenty drops not dangerous. Dr. Lallerstedt says that he has been using gelsemium for seventeen years, and that he has given as much as one hundred and twenty-five minims without the least bad effects; that he gives it to infants and in old age, and that he frequently gives forty minims for sick-headache. I agree with the editor of the "Therapeutic Gazette," who, in commenting on these statements, concludes that the fluid extract which the doctor used must have been a remarkably inert preparation. The editor also records five fatal cases in which the dose which led to collapse and death ranged all the way from ten minims to a teaspoonful.

Dr. Weir Mitchell uses gelsemium largely in his practice, and was the first to advocate its employment in efficient and increasing doses for neuralgic and spasmodic affections.

HERNIA CEREBRI.

Reference is made in the "Medical Record," March 8, 1890, to Dr. C. E. Olmsted's account of a peculiar congenital protrusion of brain-substance through the frontal bone of a child five years old. The opening in the frontal bone was about the size of a silver dollar. Pulsations in the tumor could be distinctly seen as well as felt. The child had convulsions at irregular intervals, coming on without apparent cause, at which times the brain-substance would recede through the opening in the skull, and the defect in the frontal bone could readily be defined by the finger. After the convulsion, the tumor—about the size of a plum—would again appear on the forehead.

REPORT OF AN INTERESTING CASE OF FEIGNED INSANITY.¹

By M. D. FIELD, M.D.

ON December 17, 1887, my attention was called to J. D., who was then a prisoner at the Tombs, indicted on a charge of grand larceny of the first degree. If convicted under this charge, he would have been subject to from five to ten years' imprisonment. I learned that he had been at the Tombs since November 16th, a period of a little over four weeks. During this time the keeper stated he had been in the same state in which I found him; that since admission he had never spoken a single word; and that he had maintained a perfect indifference to everything about him; that he had never made any voluntary movement, except slight opening and closing of his lips. If taken hold of, he would follow wherever led; if put in a chair, or any place, there he would remain; he would not take food or water when placed in front of him, or if left beside him; and, given an opportunity to partake when nobody was present, it was never found that he had taken advantage of such opportunity. If food was placed in his mouth, he would swallow, in a mechanical sort of way; the attendants were very confident that it was difficult for him to swallow any solid food, and they were in the habit of giving him bread soaked in soup. They never knew him to voluntarily use the pails, the only means of relief in the cells; occasionally he would pass his urine in his clothing, or in the bed; a few times they have placed him on the seat and left him for several hours, when he had had slight passages from his bowels.

¹Read at the March meeting of the New York Neurological Society.

From the closest questioning, it seems to be a fact that his secretions were very much diminished, though, of course, he was having a meagre diet, with little fluids. So thoroughly convinced were the attendants that he would make no effort to help himself in any way, they were afraid to let him lie flat in bed, lest he should smother in the pillows and bedding; they would bolster him up, in a half-sitting position, and, though asked to make frequent observations, which they claimed to have done, during the night, they reported that they never found evidences that he had moved from the exact position in which they had placed him, and that they always found him in the same position, eyes open, and with the same staring expression. They also reported that in leading him out and in from his cell, the doorway of which was low, that he would strike his head against the iron frame, unless they took pains to push his head down low enough to go under. In this way he received several pretty severe blows, before they guarded against injury in this way. In fact, he appeared to all those about the prison to be without knowledge of, or interest in, things about him, being totally indifferent.

With this history, I saw and examined him for the first time on December 17, 1887; and I saw him a number of times afterward. He was a tall, spare man; rather pale and anæmic. He was led into the examination-room by one of the keepers, in front of a chair, when the keeper shoved him back, and he sank down without resistance, and seemed to simply fall into place. From this position he never moved. He had a fixed, staring expression. Only occasionally would he wink. He could not be made to speak, make any effort, or give any evidence that he comprehended what was said to him or what was going on about him. His dress and person bore every evidence of perfect neglect. His pulse was rather small and quick; his breathing was shallow; his temperature was apparently slightly subnormal (not taken by thermometer); all reflexes, both deep and superficial, appeared normal. His limbs, if raised out of position and then released, sunk back into position as if from gravitation, but not with the dead

fall of a paralyzed limb ; there was nothing of a cataleptic nature about the case that I could observe.

I might remark here that two physicians who had seen the case had reported to the District Attorney that he was in a cataleptic, or cataleptiform, condition, and not in a condition to be tried. If I stood directly in front of the man and were to throw water in his face or prick his skin, he would give no manifestation of feeling. If I stood behind him, where he could not see me, and snapped drops of cold water from my fingers, so as to strike the side of his face, involuntary movement followed. I made firm and prolonged pressure upon the supraorbital nerve, the only response being that his face became much suffused and a few tears came from his eyes. But there was no expression of pain or anger in his face, though the pain inflicted must have been severe, and certainly was as great as I felt justified in producing.

I had repeated interviews with this man, and tried various inducements to get him to speak, but without avail. One day I had the photographs, which I will exhibit, taken. The light was poor, and the exposure made in both cases was over a minute and a half. I may remark that the photographer was astonished at the perfect quiet of the patient during that time, there being not the slightest evident movement. The distinctness with which the eyelashes are shown will indicate this. I do not believe that the man was any stiller while his photograph was being taken than during the whole of all, and every, interview that I had with him.

During his stay at the Tombs he must have lost some thirty or forty pounds in weight, and, in fact, he became very emaciated. I began to think he would starve himself to death. On inquiring into the history of this man, I learned the following :

That on or about the 7th day of November he stole a watch ; made rational effort, and did escape ; was afterward captured and taken to the 57th Street Court. Here he pled not guilty to the charge, and signed his name to the paper. That night he was reported by the prison keepers to have

made an outcry, and to have had "a fit." After that he would not speak or eat, and was stolid and indifferent; apparently in the same condition as I found him. I saw his father, who told me that when thirteen or fourteen years of age he received a fall; could give no definite injuries, but that he was laid up five or six weeks; that he had always been moody—at one time gay, at another time depressed. Family history negative. His father informed me that he had been working for his brother up to the day of his arrest; that he had never observed anything strange in his actions until he called upon him in prison. He told me that his son had been several times convicted of crime. I was informed by the police department that on July 11, 1879, he was convicted for felonious assault, and sentenced to one year in the Penitentiary. On October 16, 1880, he was arrested, charged with larceny of gold watch and chain; was found guilty, and sentenced to two years in State Prison; that on November 1, 1882, he was charged with same offence; found guilty and sentenced to six years in the State Prison; was discharged in April, 1887. Nothing was known of him from that time until the present charge. At the time of arrest, on the present charge, was perfectly rational, and made offer to return the watch, or see that it was returned, if not prosecuted. The friends of J. D. and the authorities at the Tombs became alarmed at the manner in which he was running down, and the prison officials were anxious to get rid of him; but, owing to the peculiar report of the two physicians who had examined him for the District Attorney, who stated that he was in a cataleptic condition and not fit to be tried, yet not stating that he was either sane or insane, he could not be disposed of by trial; it was a difficult problem to know what to do with him. Being convinced that the man was feigning everything, I was anxious to have him sent to Bellevue Hospital for observation. He was finally sent to Jefferson Market Prison on December 29, 1887.

After this I lost track of him until one morning I saw an article in one of the daily papers, headed "The Silent Man departs," and then learned that he had managed to

saw out a bar and made good his escape, on the night of February 5, 1888. He was aided in escaping by one John Mack, who was recaptured and sentenced to three months in the Penitentiary. The other day I had an interview with Mack, and learned that for nearly a month he had been talking with the prisoner; and that he had aided him in this deception, as he would keep watch and thus allow him to move about and get a little relaxation; if anybody approached, he would communicate with him and, of course, he would assume his old attitude.

I have reported this case on account of the interest it has been to me; not so much from the correctness of its simulating any particular form of insanity, as for the persistent maintenance of the condition assumed for so long a time. He must have lost from one-fifth to one-quarter of his entire weight by simply depriving himself, voluntarily, of food. And the wonderful power that he displayed of maintaining such a given attitude; the fixed stare being kept up constantly for weeks and even months. The manner in which he could have pain inflicted without any expresion of pain or anger was wonderful.

Considered as a type of insanity, it was very true to that form of insanity described as stuporous insanity, or acute dementia. That form of insanity frequently begins suddenly and after some shock. Had an innocent person been cast into prison, or had a guilty man been brought up short for the first time, one could easily see that the shock would be sufficient to produce that form of insanity in a predisposed subject. Catalepsy appears with that form of insanity frequently. I did not find catalepsy, but two physicians, employed by the District Attorney, had so reported. The total indifference, stolidity, refusal of food, and refusing to speak, are all characteristic of that condition; the evident diminution of the secretions could be taken as an objective indication of this condition, for it really seemed very small, even in comparison with the small amount of food taken; the small and rather quick pulse; the slow and shallow respiration; the coolness of the skin; the temperature, if anything, being lower than normal.

The question might be asked: "Was not this nearer a case of melancholia with stupor?" To this we must reply that the attitude and expression was more one of stupidity than one of depression. There is more evidence in this form of insanity of knowledge of the surroundings than in one of stuporous insanity; and it is usually believed that the attitude of one suffering from *melancholie avec stupor* is maintained by the patient because he is dominated by some delusion; and we would not expect such a sudden onset as in this case.

On the other hand, the points against this being a case of a genuine stuporous insanity seem to me to be the following:

There was a strong motive for simulation; five to ten years in prison was staring him in the face; and the fact of this being his third conviction for this offence, he was likely to get the full penalty of the law. His family history, as given by his father, showed no predisposition; his rational actions up to and shortly after his arrest, while he showed insensibility to pain and to reflex action when he could prepare himself for and use his will, yet when off his guard his reflexes were all found to be normal, his pupils being actively responsive; though he swallowed mechanically, yet the readiness with which he did take food after it was placed in his mouth, if not indicating volition, showed very good reflex action. Though his pulse was small and his hands cool and his bodily temperature probably a little subnormal, he had not the deeply congested and clammy hands so common in cases of dementia.

Again, the stuporous insanity is rarely met with in persons over twenty-five years of age. Finally, if this was not a case of stuporous insanity, acute dementia, or melancholia with stupor, it would have to be rejected as coming near any form of insanity.

A fellow prisoner who was recommitted to the Tombs, reported that he saw this man a year and a half later looking well and prosperous.

Periscope.

CRANIO-CEREBRAL TOPOGRAPHY.

By C. L. DANA, M.D.

The writer has frequently received requests for copies of the accompanying rules, originally published in the *Medical Record*, January 12, 1889, and ventures, therefore, to reproduce them here. They have been carefully revised, and condensed to some extent.

I have also added directions regarding the method of tapping the ventricles and of reaching the internal capsule.

A very good cyrtometer has been made for me by Meyrowitz Brothers, Fourth Avenue and Twenty-third Street.

RULES OF CRANIO-CEREBRAL TOPOGRAPHY.

Rule I. The longitudinal fissure.—This corresponds with the naso-occipital arc.

Rule II. The fissure of Rolando.—(a) *The upper end.* Use the cyrtometer as directed ; or, measure the distance from the glabella to the inion ; find 55.7 per cent. of this distance, and the figures obtained will indicate the distance of the upper end of the fissure of Rolando from the glabella. As the naso-occipital arc ranges from 28 to 38 ctm. (11 to 15 inches), the point sought for lies from 15.7 to 26.8 ctm. ($6\frac{1}{8}$ to $10\frac{1}{2}$ inches) from the glabella.

(b) *The course of the fissure.* Starting from the upper end of the fissure, lay off with the cyrtometer a line forming an angle of sixty-seven degrees anteriorly with the longitudinal fissure. This gives the direction of the upper two-thirds of the fissure, or for about 5.6 ctm. ($2\frac{1}{4}$ inches). The lower third, about 2.1 ctm., is slightly more vertical. The bend of the fissure is about on a level with the anterior end of the parietal fissure. The total length of the fissure averages 8.5 ctm. ($3\frac{3}{8}$ inches).

(c) *To find the lower end* more exactly, if needed : Lay off a line from the stephanion to the asterion, and another from the bregma to the external auditory meatus. The

not extend down so low in children, and is a little more vertical.

Rule III. To find the fissure of Sylvius.—Draw a vertical line from the stephanion to the middle of the zygoma. Draw a horizontal line from the external angular process to the highest part of the squamous suture; continue this back, gradually curving it up till it reaches the parietal eminence. The junction of the two lines will be at the beginning of the fissure of Sylvius. The vertical line indicates nearly the position of the anterior or vertical branch of the fissure, which is, however, directed a little more forward, and is about 2.5 ctm. (1 inch) in length. The posterior part of the line indicates the position of the posterior branch of the fissure. The triangular gyrus and motor speech-centre lies just anterior to the vertical branch of the fissure. The operculum lies just back of it. The tip of the temporal lobe reaches nearly as far forward as the posterior edge of the orbital process of the malar bone. The fissure of Sylvius is separated from the lower end of the precentral sulcus by a convolution 1 ctm. wide on the average (Horsley).

Control Measurements and Variations.—Reid's method of finding the fissure of Sylvius is to "draw a line from a point $1\frac{1}{4}$ inch behind the external angular process to a point $\frac{3}{4}$ inch below the parietal eminence. The ascending branch starts from a point $\frac{3}{4}$ inch back from the anterior end of this line, and 2 inches (5 ctm.) back of the external angular process."

Dr. Hare draws a line from the external orbital process to the inion. A point $1\frac{1}{2}$ inch behind the anterior end of this line marks the beginning of the fissure, and a straight line from here to the parietal eminence marks the course of the posterior or main branch.

The fissure of Sylvius runs nearly horizontally, and lies either under or a little above the uppermost part of the parieto-squamous suture. *This suture, the external orbital process, and the parietal eminence* are the guiding landmarks by help of which the surgeon can often operate without marking down lines on the scalp.

In children the fissure is sometimes higher and more oblique.

Rule IV. To find the parieto-occipital fissure.—Find the lambda, mark a point 3 mm. anterior to it, draw a line through this at right angles to the longitudinal fissure, extending about 2.25 ctm. (1 inch) on each side of the median line. This marks the net of the parieto-occipital fissure. If the lambda cannot be felt, its position may be found by measuring the naso-occipital arc, and taking 22.8 per cent of it. This indicates the distance of the lambda from the inion or external occipital protuberance. The average distance in male adults is 7.42 ctm. ($2\frac{7}{8}$ inches). It is greater in women, by a little over a millimetre, than in men.

Control Measurements and Variations.—The position of the fissure ranges from just under the lambda to as much as 12 mm. ($\frac{1}{2}$ inch) in front of it.

It is rather further in front, proportionately, in young children, and, according to Féré, in women.

Rule V. To find the interparietal sulcus.—First mark out the lines for the fissure of Rolando, fissure of Sylvius, and parieto-occipital fissure, and mark the position of the parietal eminence. Find a point on a level with the bend of the fissure of Rolando, and about 2 ctm. ($\frac{3}{4}$ inch) behind it. From this draw a curved line up and back, keeping it half-way between the fissure of Rolando and parietal eminence as it ascends, and half-way between the parietal eminence and longitudinal fissure as it passes back. Continue the line back till it reaches a point just outside the external end of the parieto-occipital fissure. This fissure divides the parietal lobe into a superior and inferior lobule. The parietal eminence lies over or a little behind the supra-marginal gyrus, and about over the middle of the inferior parietal lobule.

Control Measurements.—This fissure has a most variable arrangement, and no absolute rule can be laid down.

Its anterior inferior end is about an inch from the angle formed by the prolongation of the fissures of Rolando and Sylvius.

Rule VI. To find the inferior precentral or vertical sulcus, and the inferior frontal and superior frontal sulci.—The

inferior precentral or vertical sulcus passes nearly vertically just posterior to the coronal suture. Its lower end is 1 ctm. above the Sylvian fissure (Horsley), and 2 mm. behind the coronal suture. Its upper end reaches to the level of the mid-point of the fissure of Rolando, and is 4 mm. behind the coronal suture. It lies 2 to 2.5 ctm. anterior to the fissure of Rolando.

The inferior, or second, frontal sulcus passes forward from the precentral sulcus at a point a little above the stephanion. It continues forward in a line nearly identical with the frontal part of the temporal ridge (Reid).

The superior, or first, frontal sulcus begins at a point half-way between the fissure of Rolando and a line prolonged up from the inferior precentral sulcus (Horsley).

This point should be from 2 to 2.5 ctm. in front of the fissure of Rolando. The fissure passes forward parallel to the longitudinal fissure, and its line, if prolonged, ends in the supra-orbital notch (Reid).

Rule VII. To outline the frontal lobes.—The anterior end of the frontal lobes reaches to a point determined by the thickness of the frontal bone. This ranges from 2 to 8 or more mm. $\frac{1}{12}$ to ($\frac{1}{3}$ in.).

The floor of the anterior fossa reaches in front to a level a little above the supra-orbital margin (16 mm., $\frac{3}{4}$ in., Hefstler). It slopes down and backward, its posterior limit being indicated by the lower end of the coronal suture.

Rule VIII. To find the temporal lobe and the first and second temporal sulci.—The temporal lobe is limited above by the fissure of Sylvius, below by the contour line of the lower border of the cerebrum. This corresponds to a line drawn from a point slightly (about 12 mm.) above the zygoma and the external auditory meatus to the asterion, and continued on along the superior occipital curve to the inion. The anterior border of the lobe corresponds to the posterior border of the orbital process of the malar bone. The posterior border of the temporal lobe is somewhat arbitrarily found by drawing a line from the Sylvian fissure line at a point 2.5 ctm. below the parietal eminence, backward and downward to the anterior occipital fissure.

The temporal lobe is about 4 ctm. ($1\frac{1}{8}$ inch wide) at the external auditory meatus. A trephine, as Bergmann states, placed half an inch above the meatus would enter the lower part of the lobe. The middle of the lobe is in a vertical line from the posterior border of the mastoid process. A line from the upper end of the fissure of Rolando to the point of the process would pass through this important sensory area.

A point just over the posterior part of the first temporal gyrus is found (Barker) by drawing a line $1\frac{1}{2}$ inch long horizontally back from the external meatus, and then erecting a vertical $1\frac{1}{2}$ inch. At this point the skull is sometimes trephined in mastoid disease.

The first temporal gyrus is about 1 inch (2.5 ctm.) wide; the second temporal is a little narrower (Reid).

Rule IX. To find the occipital lobe and anterior occipital fissure.—The upper anterior border lies under a line drawn from just above the lambda (1 to mm.), curving out and down to a point about at the junction of the anterior and middle third of the line from the inion to the asterion. The lower border corresponds pretty closely to the superior occipital curved line. The anterior occipital sulcus when present, should lie in the anterior border of the lobe.

Rule X. To find the position of the central ganglia, viz., corpus striatum and optic thalamus, draw a line from the upper end of the fissure of Rolando to the asterion, practically a vertical line. This limits the optic thalamus posteriorly. A vertical line parallel to the first, a little in front of the beginning of the fissure of Sylvius, limits the corpus striatum anteriorly. A horizontal plane 45 mm. ($1\frac{3}{4}$ inch.) below the surface of the scalp at the bregma, limits the ganglia superiorly. The ganglia lie about 35 mm. ($1\frac{3}{8}$ inch) below the superior convex surface of the brain (Féré).

Rule XI. To reach the internal capsule (in its anterior part), and the common seat of cerebral hæmorrhage.—Dr. C. K. Mills suggests trephining over the temporal lobe posteriorly and low down, then passing the exploratory-needle forwards and inwards.

A better way, according to experiments made by myself, is to find the mid point between the extremities of the basal ganglia (*vide* Rule XI.). Then trephine, at a point about 3 ctm. ($1\frac{1}{4}$ inch) from the median line, and plunge the needle directly down and slightly outward, for a distance of 4 to 6 ctm. ($1\frac{1}{2}$ to $2\frac{1}{4}$ inches).

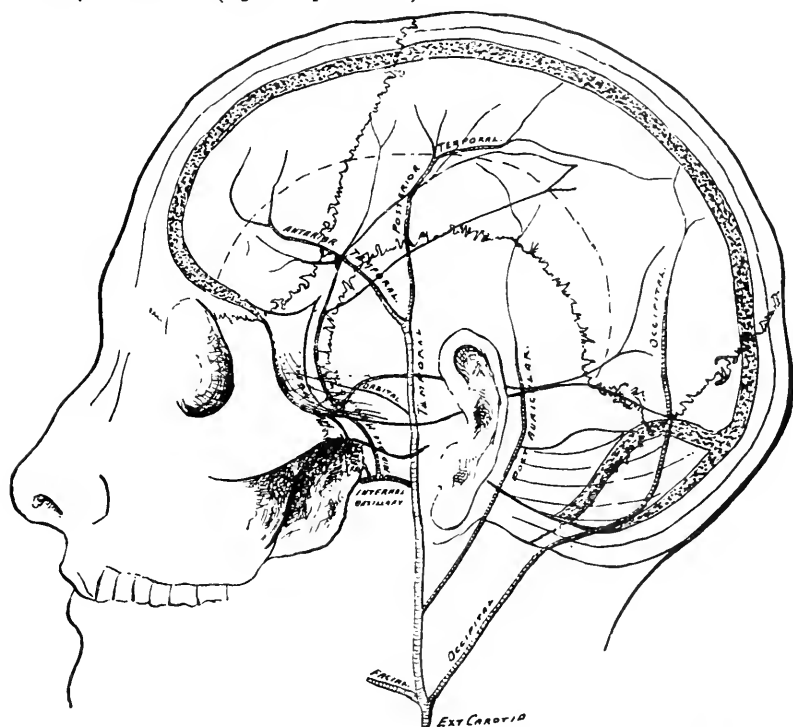


FIG. 2.—Showing the Course of the Arteries of the Scalp and Dura Mata.

Rule XII. To reach the lateral ventricles.—A number of routes may be taken. The lateral is recommended by Keen. Mark a point $1\frac{1}{4}$ inch behind the external auditory meatus, and $1\frac{1}{4}$ inch above a base line made by drawing a line through the lower border of the orbit and the external auditory meatus.

Trephine at this point and plunge the director into the brain in the direction of a point $2\frac{1}{2}$ to 3 inches vertically above the opposite external meatus.

The ventricle lies at a depth of 2 to $2\frac{1}{4}$ inches (5 to 5.7 ctm.).

Rule XIII. To avoid the meningeal arteries and central sinuses.—The course of the middle meningeal artery has been described and is seen in Fig. 2. This artery is the only one of importance or very definite course. The superior longitudinal sinus generally (not always) lies a little to the left of the median line. The torcular Herophili lies approximately under the inion. The lateral sinus lies generally under the line from the inion to the asterion, and just grooves the postero-inferior angle of the parietal bone.

Rule XIV To outline the base of the brain.—The rules for this are given in Rules VII., VIII., and IX., for finding the frontal, temporal, and occipital lobes.



FIG. 3.—Showing the Relations of the Cranial Surface to the Convolution and Cortical Centres.

Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, Tuesday Evening, April 1, 1890.

The President, Dr. GEO. W. JACOBY, in the chair.

Dr. M. D. FIELD presented a report of a

CASE OF FEIGNED INSANITY. (See p. 401.)

Dr. FITCH said that malingerers could not feign mania and melancholia so successfully as they could the condition of stupor. It was difficult for examiners to determine the exact nature of the case, when the latter state was simulated, for obvious reasons. He related some instances of feigned insanity that had come under his observation.

Dr. INGRAM referred to a case described by Esquinol which was similar in many respects to that of Dr. Field.

Dr. SACHS recalled an interesting case that he had observed in Westphal's clinic nine years ago. The motive of the patient was to escape military duty. The man was twenty-two years of age. He became suddenly mute for six or seven months. There were no other symptoms. All the military physicians had either agreed that the man was insane or suspected that condition. He was sent to Prof. Westphal. At this time he further simulated a contracture of the right leg. It was impossible to surprise him at an unguarded moment, for even at night they would invariably find the contracture present. He would so envelop the limb in the bedclothes that any attempt at examination would awaken him. Finally an officer was hidden in a room where the patient was to meet a friend, with whom he talked quite freely, and the malingering was thus discovered.

Dr. FISHER thought it would be impossible for any one to say whether a person was insane or not when in a condition such as had been described by Dr. Field, unless the person were under constant observation. Simulators seemed to be more often found in the classes of feeble-minded and imbecile, or in those with hereditary neurotic taint.

Dr. LESZYNSKY related an interesting case of deception practised by a woman with chronic mania. She inserted a piece of glass into her arm, which one of the physicians in the asylum removed by operation. She then claimed that there was another piece in a neighboring spot, and this too was located and removed. This was repeated over and over again quite a number of times before it was ascertained

that she was herself inserting the pieces of glass into her flesh.

Dr. DANA had seen stuporous forms of insanity, at Bellevue, often associated with catalepsy. They were a species of katatonia. In cases of simulated catalepsy there was an excellent test which he had made use of to discover the simulation. It consisted in placing the supposed cataleptic before another patient in the familiar attitude with his fingers to his nose. This position appeals to the sense of the ridiculous to such an extent that the simulator will finally break down, as a rule; of course, the true cataleptic remains unaffected.

Dr. G. M. HAMMOND then read a paper upon

THE RATIONAL TREATMENT OF SCIATICA. (See p. 333.)

Dr. DANA thought that the author of the paper had done good service in calling attention to the fact that rheumatism, gout, and syphilis were not causative elements in sciatica, but merely diatheses, at times coincident with the disease of the nerve. Almost all cases were of an inflammatory nature. There was a minority of cases, however, in which there was no actual neuritis, but a pure neuralgia, often reflex and due to pelvic irritation, and especially found in young women. He believed the treatment outlined to be rational. Rest was fundamental, but he had had quite as much success with strong counter-irritation, in addition to the rest, as with the application of cold.

Dr. STARR mentioned the fact that acupuncture had been employed as a remedy in sciatica, a contributor to the "Practitioner" having recently called attention to the method, claiming that fluid accumulating in the sheath of the nerve might thus be evacuated. He had himself had no experience with it.

THE PRESIDENT differed from the author of the paper and from Dr. Dana in their statement that rheumatism, gout, and syphilis had no particular causative relation to sciatica. There was not perhaps so much relation between sciatica and gout and syphilis as between sciatica and rheumatism, but his experience led him to be convinced of such relation. He had also met with a number of cases of sciatica due to diabetes; and if diabetes could do so, why could not the rheumatic poison produce a similar sciatic neuritis?

In treatment he had found the galvanic current very unsatisfactory, but the application of cold useful. He had also employed ichthyol with considerable benefit.

The following officers were elected for the ensuing year :

President—Dr. Landon Carter Gray.

First Vice-President—Dr. B. Sachs.

Second Vice-President—Dr. E. D. Fisher.

Recording Secretary—Dr. Frederick Peterson.

Corresponding Secretary—Dr. W. M. Leszynsky.

Treasurer—Dr. Græme M. Hammond.

Councilors—Dr. G. W. Jacoby, Dr. C. L. Dana, Dr. M.

D. Field, Dr. M. Allen Starr, Dr. E. C. Seguin.

FREDERICK PETERSON,

Recording Secretary.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, April 28, 1890.

The President, Dr. S. Weir Mitchell, in the chair.

Paper of Dr. Bassette.

DISCUSSION.

Dr. S. WEIR MITCHELL.—I was probably the first to suggest the use of gelsemium in these affections. I used it in a number of cases, and my results were reported in some clinical papers published ten or twelve years ago. At that time I recommended that it be pushed to an extreme limit until the toxic effects were obtained. I have recently observed some interesting results from the use of this drug. In two cases there has been complete cure of spasm of the rotatory muscles of the neck. In a recent case at the Infirmary for Nervous Diseases great relief has been afforded, although the cure is not complete. In another case, on which an operation was about to be performed, I recommended to Dr. M. Roberts the use of gelsemium in full doses. The relief was so great that the patient declined operation.

Dr. Bassette speaks of spasm of the spinal accessory nerve. I am certain that very few of the rotatory spasms of the neck are pure spinal accessory spasms. I have not seen any case where section of the spinal accessory nerve caused a spasm of the neck-muscles to stop for more than twenty-four hours. I have seen the muscle cut, the nerve divided, and the nerve divided and stretched, but I have not seen any permanent benefit. The reason for this failure is that the rotating muscles of the neck are situated on both

sides. For example, the right sterno-cleido muscle turns the head to the left, while certain muscles on the left side of the neck at the back also aid to rotate the head to the left, both groups of muscles being combined for rotatory movement of the neck. Undoubtedly there are, within the brain, centres for rotation of the head, and these centres control muscles on both sides. This makes it impossible to expect good results from the division of muscles on one side of the neck.

Dr. CHARLES K. MILLS.—Dr. Bassette has called attention to the fact that patients suffering from these local spasms will often endure large doses of the drug for a long time even when a good preparation is used. It seems to me that in these cases of local spasm there is a resistance to the action of motor depressants similar to the resistance to narcotics seen in sensory disturbances.

One argument against operation is that these diseases are due to irritation of the cerebral motor centres; and these centres govern movements, and not muscles. Another reason against operation, as I have pointed out in an article in the "University Magazine," are the extremely extensive and peculiar connections of both motor and sensory nerves about the spinal accessory down to the fourth or fifth cervical segment. The close connection between the occipitalis, major and minor nerves, and the spinal accessory is one explanation of the failure of operation.

Dr. Bassette has wisely pointed out the great variations in the effect produced by this drug. I would supplement her remarks by expressing the hope that we might soon reach some responsible standard preparations, especially of the more potent agencies, wherefrom we may at least expect uniform results in the same individual and under similar circumstances. About the only trustworthy method of getting real effects from many medicines is, by gradually increasing the dose, the characteristic phenomena are exhibited, oftentimes a tedious procedure and fraught with painful delays to the sufferer.

Among the analgesics, tinctures are practically out of the question, and fluid extracts only to be used. Of these the qualities differ, so that I am now using, whenever possible, the normal liquids of Parke, Davis & Co., and with very gratifying results. Doubtless other firms can make as good, but so far as I know have not yet done so.

A very valuable effect of the drug gelsemium is to allay spasmodic cough, and has given me most comforting aid in the coughs complicating the recent influenzas. This, added to expectant mixtures, immensely improves them.

SPINAL CARIES AND PACHYMEINGITIS INVOLVING DORSAL AND CAUDA-EQUINAL REGION.

By WHARTON SINKLER, M.D.

No satisfactory history of the case could be obtained, as the woman was demented and none of her friends were ever seen. The woman had two prominences of the spinal column—one occupying the entire lumbar region, the other the upper dorsal.

The patient was helpless, both legs and arms being paralyzed. The legs were flexed upon the thighs, and the thighs upon the abdomen. These contractures were probably of long standing; the tendons were contracted, the muscles much wasted, and adhesions had formed in the joints.

The reflexes were exaggerated. Sensation was not much impaired. She had incontinence of urine.

Post-Mortem.—At the base of the right lung anteriorly a cheesy nodule, about the size of a hickory-nut, was found. The upper lobe of the left lung was infiltrated with cheesy nodules. Both pleuræ were adherent throughout. In the anterior portion of the spinal canal, at the seats of the curvatures, the vertebræ were eroded, and cheesy deposits were found adherent to the dura and pressing upon the cord. The dura was thickened at these places. The cord is being hardened for microscopic examination. No other lesions were found.

CYST OF THE LENTICULAR NUCLEUS.

By WHARTON SINKLER, M.D.

B. H., aged sixty-one, occupation a miner.

May 24, 1888: Family history negative. The patient has enjoyed good health until the present attack. Denies specific history. Six years ago the right leg became suddenly paralyzed, and this soon extended to the right arm and in succession to the left leg and arm. There is no history of injury of any kind. He was unable to speak for a short time after the onset of the paralysis, but he soon regained his speech. His paralysis has also improved, and he is able to walk on crutches.

August 17, 1889: Patient is quite unable to walk. There is but little motor power remaining in the lower extremities, and there is considerable loss of power in the upper extremities. Speech is impaired, being slow, embarrassed, and somewhat scanning, at the same time indistinct. There is a tendency to ready laughter. The patient does not believe that things appear ludicrous, but the laughter is only a response to the muscular movements of the face. There is dribbling of saliva. The knee-jerk is exaggerated on both sides, though there is some contracture of the knee-tendons. There is also some rigidity of the left arm. The pupils are equal, regular, and responsive to light. There is no headache, no pain; sleep is good. Eyesight and memory are thought to be impaired, but hearing is unimpaired. There is incontinence of urine.

April 20, 1890: On the afternoon of April 17th the patient had a chill, followed by temperature of 105° . From this time until he died, on the 20th, his temperature fluctuated irregularly between normal and $105\frac{1}{2}^{\circ}$, and his pulse ranged from 108 to 150. Shortly after the onset of the attack he vomited several times. He suffered no pain, and said he was feeling good. Examination of the thorax and abdomen proved negative until shortly before death, when signs of pulmonary congestion were present.

Post-Mortem.—Brain: In the right hemisphere an old hemorrhagic cyst, about five-eighths of an inch in length and three-eighths of an inch in width, was found involving the outer half of the lenticular nucleus about one-third of an inch back of its head. Anteriorly it was bounded by softened tissue involving to a slight extent the internal capsule. Except at this one point, it was almost entirely limited to the lenticular nucleus. The internal capsule was not affected. The vessels at the base were atheromatous.

The cord was not examined.

Both lungs were much congested at their bases posteriorly.

No other lesions were found.

The section of the brain was made in all directions, but no lesions found to explain the paralysis.

DISCUSSION.

Dr. CHARLES K. MILLS.—The first specimen is of interest in regard to the possibility of operation. You have two isolated lesions and the cord itself has not suffered much at either place. It seems to me that if these lesions could

have been recognized, and MacEwen's operation of removing the posterior arches, and allowing the cord to expand, had been performed, it is possible that this woman would have been saved some suffering. Even the lesion in the cauda-equal region is a membranous one, and was probably operable.

Dr. JAMES HENDRIE LLOYD.—In the report of a series of cases of spinal caries with autopsies, made a year ago to the College of Physicians, I attempted to demonstrate the reason why in many of these cases we have paralysis of motion without the involvement of sensation. In most of the autopsies that I have seen, in which there has been paralysis of motion without paralysis of sensation, there has been an angular curvature of the spine, causing a doubling in of the lateral columns, but allowing the posterior columns to escape almost entirely. This seemed to me to be a satisfactory anatomical explanation of the condition usually found. A recent writer explains this in a different way, and attributes it to a peculiar distribution of the blood-supply of the cord. The case of Dr. Sinkler's, in which there is very little anatomical lesion, would seem to point to the possibility of some interference with the blood-supply of the anterior part of the cord as the cause.

In regard to operation: In most of these cases there is considerable thickening of the theca and such deposit of fibrinous matter extending down the spinal canal that I think the outlook for operation is not as flattering as it has been considered by some who have had but one or two cases on which to base their conclusions.

Dr. FRANCIS X. DERCUM.—The fact pointed out by Elliot, that the cord shows actual disease only very late in these cases, is a very important one, and it indicates, to my mind, that in a certain percentage of cases, after other measures fail, operation should be considered. If the cord is not diseased, and we can give it more room, benefit should follow. I recognize that, in the old and long-standing cases, operation is out of the question.

Dr. WHARTON SINKLER.—In conclusion, I wish simply to remark that this specimen of the cord demonstrates how extension may be of service in these cases of caries with pressure on the cord of long standing: We see here that the cheesy and tubercular deposits are limited to the dura mater and have not involved the cord, nor is there any adhesion between the dura and the cord at the seat of deposit. Stretching may modify the relation of the cord to the dura at these points, and, by relieving the pressure on the vessels, benefit the nutrition of the cord.

Book Reviews.

A NEW MEDICAL DICTIONARY: Including all the Words and Phrases used in Medicine, with their Proper Pronunciation and Definitions. By Geo. M. Gould, B.A., M.D. Philadelphia, 1890: P. Blakiston, Son & Co. Small octavo, 520 pages. Half dark leather, \$3.25; with thumb-index, half morocco, marble edges, \$4.25.

It is an era for coinage of new words, and it seems as though this year were one for dictionaries, of every description and serving all kinds of etymological purposes. When one can obtain a large Unabridged Webster's Dictionary as a premium for a subscription to a magazine, or one dollar in money, there is hardly an excuse to be without a friend—no matter if the make-up is poor—in case of an etymological necessity.

There are also the already well-known Century, Appletons', and National Medical Dictionaries, of broader scientific character, handsomely arranged and illustrated, and so profusely descriptive as to be veritable encyclopædias. All of these serve purposes chiefly outside of the field of medicine, and therefore permit the work under consideration to fit in without really competition. There is a decided need for a comprehensive and compact Medical Dictionary. This one is to be fully appreciated by the physician and student anxious for quick information upon some perplexing and recent medical term.

It is not a mere compilation from other dictionaries, but contains the product of patient research through the various periodicals and text-books in various branches of medicine, and also contains valuable tables of abbreviations, arteries, nerves, ganglia, bacilli micrococci, ptomaines, etc. : and all is bound in a handy volume, indexed and arranged as a book to work with quickly. It will meet with success and appreciative purchasers.

PRACTICAL PHOTO-MICROGRAPHY. By Andrew Pringle, F.R.M.S. New York, 1890: Scovill & Adams. Pages 183, with six plates.

Unquestionably practical photo-micrography would be of great interest and utility to the neurologist, and particularly to the neuro-anatomist and pathologist, if he could feel reasonably certain of securing even a tolerable photograph of his specimens without devoting weeks or even months of time to it. If photographs could be produced which would illustrate the common pathological changes which take place in the cerebro-spinal system, and show them as the microscopist sees them, photo-micrography would soon supersede any other method of illustration. It has been stated many times that "photographs do not lie." It is also claimed, whether justly or not, that drawings of microscopical specimens are not always open to the same criticism. Therefore, if it were possible, if from no other reason than this, to produce good photo-micrographs, even at the expense of considerable time and trouble, photo-micrography would soon be generally employed.

Until the methods of producing photo-micrographs have been very much more simplified, and made less expensive, the practice of the art is only likely to be indulged in by the few who have unlimited time and ample means at their disposal.

It is perhaps a comparatively simple process to photograph a microscopical specimen of a fly's foot, or the various bacilli, or a transverse section of the spinal cord, but it is an entirely different matter to so clearly depict minute pathological changes that the observer can exclaim at once, on viewing the photographer's work: "This is a photograph illustrating posterior spinal sclerosis, and this is gliomatous infiltration of the spinal cord, and this is general paresis." This, as far as the reviewer's knowledge goes, has never been satisfactorily accomplished.

The novice, or he who thinks good photo-micrographs can be made with facility by any one who possesses a microscope and a camera, is soon undeceived, and is perhaps somewhat appalled, by reading this work. He will soon find that a great deal of fine apparatus is necessary, and that a thoroughly practical knowledge of illumination, objectives, chromatic and apochromatic lenses, eyepieces, and reflectors is absolutely essential if ultimate success is to be achieved.

The earnest student in photo-micrography will find Mr. Pringle's volume very valuable. It deals with its subject in a thoroughly scientific and yet practical manner. The description of all the various pieces of apparatus and how to use them, the selection of plates and the various solutions for developing them, are set forth in a clear and comprehensible manner.

The chapter on "Progressive Examples," in which the operations for subjects presenting various degrees of difficulty are detailed, is very interesting and instructive, and contains many valuable suggestions which the amateur photographer will find it to his advantage to carefully peruse.

Color-correct photography is a subject to which the author pays special attention. It is a subject of the greatest importance to the neuro-anatomist. Pathological specimens must be stained in order that their most salient features may be clearly depicted. The stains most favored are hæmatoxylin and carmine on eosine. Sections, when stained in these solutions and then photographed on ordinary plates, are exceedingly unsatisfactory, the resulting print appearing of a homogeneous blackness. This defect can be overcome, the author claims, by the use of ortho-chromatic plates.

The work, as a whole, is the best and most sensible contribution to photo-micrography that has appeared in many years. H.

BOOKS RECEIVED.

PHYSICIANS' AND STUDENTS' READY REFERENCE SERIES,
No. 5. Second Edition.

ELECTRICITY IN THE DISEASES OF WOMEN, with Special Reference to Application of Strong Currents. By G. Belton Massey, M.D. Physicians' and Students' Ready Reference Series, No. 5. Second Edition. Philadelphia, 1890: F. A. Davis, Publisher.

AN EXPERIMENTAL STUDY OF LESIONS, arising from Severe Concussions. By B. A. Watson, A.M., M.D. Philadelphia, 1890: P. Blakiston, Son & Co.

Miscellany.

NOTICE.

AMERICAN NEUROLOGICAL ASSOCIATION.

The Council of the American Neurological Association announces that the Sixteenth Annual Meeting of the Association will be held at Philadelphia, Pa., on Wednesday, Thursday and Friday, June 4th, 5th and 6th, 1890, at the Hall of the College of Physicians, S. E. corner of 13th and Locust Streets.

PRELIMINARY PROGRAMME.

Drs. S. Weir Mitchell and C. H. Burr, of Philadelphia—Spinal Chorea.

Dr. J. P. Putnam, of Boston—I. Cases of Postero-Lateral Sclerosis, with specimens; reported with special reference to the Etiology of the Disease. II. Report on a large number of cases of Multiple Neuritis, occurring among seafaring men in Northern Latitudes.

Dr. C. L. Dana, of New York—I. Anterior Myelomalacia, with specimens. II. Ingravescient Apoplexy.

Dr. G. M. Hammond—Path. Anat. findings in the original case on which Dr. W. A. Hammond's description of Athetosis was based. (Supplemented by report on cases by Dr. E. C. Seguin and Dr. E. C. Spitzka.)

Dr. G. L. Walton, of Boston—Contribution to the Study of the Traumatic Neuro-Psychoses.

Dr. F. X. Dercum, of Philadelphia—Lesion of the Pulvinar, with special reference to Wernicke's pupil-reaction.

Dr. N. E. Brill, of New York—Partial Fracture of the Cervical Spine of Twenty-two Years Standing. A contribution to the physiology of the cord.

Drs. F. X. Dercum and F. W. White, of Philadelphia—Case of Paraplegia, etc., relieved by trephining the upper dorsal vertebral arches.

Dr. B. Sachs, of New York—I. Tumor of the Corp. Quadrigem, with special reference to the Oculo-Motor Innervations. II. Crus Lesion. III. Preliminary Report on the Brains and Cords of two cases of Paralysis Agitans.

Dr. E. C. Spitzka, of New York—Limited Focal Lesion of the Pons, with Associated Eye-movement of Paralysis.

Dr. Phillip Coombs Knapp, of Boston—Brain Surgery in Relation to the Localization of Cortical Sensory Centres.

Dr. Irving Rosse, of Washington—Clinical Evidence of the Borderland of Insanity.

Dr. F. Peterson, of New York—Association of Locomotor Ataxia with Complete Nuclear Ophthalmoplegia and Muscular Atrophies.

Dr. C. K. Mills, of Philadelphia—Some Sources of Error in Trephining.

Dr. H. M. Bannister, of Kankakee—Focal Hemorrhagic Lesion of Anterior Cerebellar Crus. (Tegmenta Brachium or "Bindearm.")

Dr. B. G. Wilder, of Ithaca—I. Demonstration of the Brain of Professor Chauncey Dwight. (In connection, the brains of other distinguished scientists will be exhibited.) II. The Brain of a Chimpanzee. (In connection with which the brains of other Anthropoid Apes will be shown.)

Dr. S. G. Weber, of Boston—Hysteria and Hystero-Epilepsy Treated by Hypnotism.

Dr. J. T. Eskridge, of Denver—Acute Myelitis with Optic Nerve Atrophy.

Dr. Henry S. Upson, of Cleveland—A Third Paper on Gold Chloride as a Staining Agent for Nerve Tissue.

Dr. William N. Bullard—Diffuse Cortical Sclerosis of the Brain in Children.

Dr. James Hendrie Lloyd, of Philadelphia—Diphtheritic Paralysis, with Special Reference to Treatment.

Dr. Wharton Sinkler, of Philadelphia—A Case of Insular Sclerosis, in which an Attack of Cerebral Hemorrhage Arrested the Tremor on the Hemiplegic Side.

PROPOSAL FOR MEMBERSHIP.

Active.

Drs. Horatio C. Wood, J. Madison Taylor, Guy Hinsdale, Thomas J. Mays, Edward T. Richert and Morris J. Lewis, of Philadelphia; Dr. John Amory Jeffries, of Boston; Dr.

William C. Kraus, of Buffalo; Dr. J. K. Thatcher, of New Haven; Drs. J. Arthur Booth, Chas. Henry Brown, Richard Mollenhauer, Christian A. Herter and Mathew D. Field, of New York; Dr. James H. McBride, of Wanwatosa.

Associate.

Dr. Eugene Du Puy, of Paris, France.

A reception will be given by the Philadelphia Neurological Society to the American Neurological Association at the Art Club on Wednesday evening, June 4th, from 8 to 11 o'clock.

GREEME M. HAMMOND, M.D.,

Secretary and Treasurer.

58 West 45th Street.

Prof. William James, of Harvard University, Cambridge, Mass., in the capacity of Committee on Census of Hallucinations, of the Society for Psychical Research, desires some important data in reference to hallucinations, and will be glad to send blanks for this purpose.

The following question, especially, is desired to be answered *Yes* or *No*: "*Have you ever, when completely awake, had a vivid impression of seeing or being touched by a living being or inanimate object, or of hearing a voice; which impression, so far as you could discover, was not due to any external physical cause?*"

They who read medical journals to the exclusion of the advertisements make a serious practical mistake. It is seldom that their perusal does not prompt the use of some forgotten remedy of value, reveals an address mislaid, or the superiority of some instrument.

The Journal's advertisements ARE GREAT REMINDERS.

They make most journals a possibility, and it is certainly an advertiser's due that their various notices shall be read.

It is most urgently requested that the personal friends of this journal will aid its advertisers and endeavor to make them feel that their support is appreciated.

WANTED.—Complete file of the JOURNAL OF NERVOUS AND MENTAL DISEASE, bound or unbound, to 1888.

Special numbers wanted: January, 1888; March, 1889; January, 1890.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

UNUSUAL CASES OF CHOREA, POSSIBLY
INVOLVING THE SPINAL CORD.¹

By S. WEIR MITCHELL, M. D.

AND

CHARLES W. BURR, M. D.

J. S., male, white, aged 19, presented himself at Dr. Mitchell's clinic at the Nervous Infirmity, November 23d, 1889, complaining of constant, involuntary movements of the legs, arms and head. His maternal grandmother suffered from chorea for many years, not from birth, but while affected gave birth to the patient's mother, who was choreic from birth till death. She was always sickly. She had three children, of whom one was born dead, and another died when about a month old. The patient alone survived. She, herself, died of phthisis at the age of thirty-five. The father, a hard working man, of no bad habits, except occasionally too free indulgence in liquor, also died of phthisis. There is no history of other cases of chorea, nor of other nervous diseases in the family.

The patient was born at term; was a puny infant, and was breast-fed. He developed slowly, not beginning to walk till between two and three years of age, nor to talk until his fourth year. There is no mental deficiency. His emotional nature is highly developed. He is peevish, and at times has attacks of extreme sulkiness. He had measles when six years old, but no other serious illness. All treatment has been without influence. His aunt, a woman of sufficient intelligence to notice, says that the choreic movements began in early infancy, probably from birth.

¹ Read before the American Neurological Association, June, 1890.

Present State.—A fairly built young man of good strength, and weighing 130 lbs. Appetite and digestion are good; bowels regular, and sleep undisturbed. Thoracic and abdominal viscera show no signs of disease. K. J. is increased on both sides, 7 right and 9 inches left. The cremasteric, sole and abdominal reflexes are marked. Ankle clonus is occasionally present. At times there is a rigidity at the knee and the feet are then turned inward at the ankle. All of the above are increased by emotion, and by the administration of moderate doses of strychnia. In the right hand the dynamometer registers 140, and in the left 100°. Sensation to touch, pain and temperature is normal. So also is station.

While awake, his entire voluntary muscular system is more or less in action. The corners of the mouth twitch, the eyebrows are raised and lowered, the head turned from side to side, the shoulders shrug, the arms and legs move hither and thither. The muscles of the thorax and abdomen act irregularly in respiration. Speech is somewhat hesitating, as if he could not control the vocal muscles. Motion and emotion greatly increase the movements. For example, if told to write, at the beginning he will do quite well, but soon the writing becomes an illegible scrawl, and the arms and legs are jerked wildly about.

Again, if he is suddenly brought in the presence of a stranger, the motions are increased. During sleep he is perfectly quiet. There is no tenderness over the spine. The urine is normal. His eyes were examined by Dr. De Schweinitz, and showed the following condition: Vision=1. In each eye oval, healthy nerves, and retinas normal. Internal recti insufficient 4°. At times slight convergent squint, but diplopia absent. Expression anxious. Electrical reaction of muscles normal.

CASE II. W. L., male, white, married, aged 46, machinist.

When patient's father was about 45 years old, he began to suffer from a spasm of the legs, occurring only when in bed. The legs would be flexed on the thighs, and the thighs on the abdomen, and then the whole would be again extended. No pain, but sleeplessness. On walking, movements would cease. The legs only were affected. The disease persisted until death, which occurred in his sixty-second year. The cause of death was "enlargement of the liver."

The patient's mother was healthy. He has had eight brothers and sisters, of whom three are dead. One brother (an account of whom is given later) has an affection similar to that of the father.

Patient had ague twenty-four years ago. No other serious illness. No rheumatism. He has had five children, of whom three are living. He has never used tobacco nor has he indulged in excessive venery. No syphilis.

The present trouble began in his twenty-first year. He first noticed that when in bed his head would be suddenly jerked to the left shoulder, and a little backward. This would happen several times in the night. The muscles of the neck were sore and tired. About ten years ago he noticed irregular movements in the abdominal muscles, and jerking of the right arm. The affection has continued ever since.

Present State.—General aspect is good. Weight 190 lbs. Digestion, appetite, bowels, sleep and intelligence all normal. Temper, bad. Expression, anxious. When sitting at ease the right shoulder drops. His head is drawn suddenly back; chin pulled a little up and to the left, and the right ear approaches the right shoulder. Pain in the right sterno-mastoid. Painful spots on the outer side of the right clavicle, and above the scapula. All the muscles at the back of the neck are stiff. Both sterno-mastoid muscles are enlarged and hard. Circumference of the neck formerly 14 inches, now 19 inches. Spine curved, convexity to the left.

The right hand is alternately flexed and extended on the wrist; sometimes 12 times in five seconds; sometimes only occasionally and, at rare intervals is perfectly quiet. This movement is communicated to the entire arm. Extension of the arm increases the movements. He cannot carry a glass of water to his lips without spilling it, and often drops the glass. On the other hand, he can hammer well holding the chisel in the left hand, and never make a mistake. When writing, the elbow is held high in the air, the wrist back of the little finger is pressed hard on the table, the fingers are squeezed together. The first few words can be read, but soon a scrawl is produced. The abdominal muscles are also affected. The legs and left arm are free. K. J. is slight, but very reinforcible. No clonus. Dynamometer, R. 135; L. 135. Sensation normal. Some numbness in fingers of right hand. There is also working of the eyes and frowning. Electrical reaction normal.

CASE III. J. L., brother of W. L., aged 52, truck farmer, married, two children.

When a child had Pott's disease so badly that he became paraplegic. He recovered sufficiently to walk. No other illness.

Present trouble began twenty years ago with sudden, but not rapid, flexion and extension of the legs and thighs. At the same time there were cramp pains in the calves, which have continued ever since. The movements occur only when sitting or lying, either on the back or side, and are worse before, but not during, a storm. No trouble in walking, except that on starting there is a slight rigidity in the knee. Similar movements of flexion and extension of the forearms began about seven years later, without pain.

For the last two years he has been unable to retain his urine. Bowels are loose, but can be controlled.

Present State.—While observed, patient was quiet. Patient is a thin, wiry man. Kyphosis in the mid-dorsal region very marked. No palsy, no wasting. K. J. O. but reinforcing. No plantar nor cremasteric reflex. Healed sinus on the anterior aspect of thigh and on the inner aspect. Dynamometer, R. 180; L. 180. Station good with eyes shut or open. Sensation good. No rigidity. General health good. Emotion has no effects upon the movements.

The first of these cases is extremely rare. It is certainly very unlike the common chorea of childhood, which, under proper treatment, is recovered from in a few weeks or months; and is also unlike the cases of Huntington in being congenital and in the absence of marked mental symptoms.

That organic changes are present somewhere in the motor tract of the patient, may, we think, be admitted, because of the extreme chronicity of the affection, its resistance to all treatment, and the presence of very distinct ankle clonus and rigidity, these latter symptoms pointing to the involvement of the spinal cord. Whether, however, the changes are confined to the cord is a more difficult problem to solve. In the dog, in whom the disease is very similar to that found in this man, section of the cord does not stop the movements, and in it are found, except in the beginning of the trouble, organic changes.

In the patient, too, the movements bear a certain resemblance to those in dogs. This, however, is admittedly not a very strong argument. The involvement of the face seems to be against the view that the trouble is simply spinal.

In the second and third cases it is, of course, impossible

to determine whether the father had really choreic movements. Further, the inheritance has been only general and not specific, for the patients were born before the father was affected.

In the second case the chorea is complicated by spasmodic wry neck. Indeed, one might say that it was simply one of those cases of spasmodic torticollis in which the spasm has extended to other muscles, were it not that the movements are distinctly choreic.

As to its morbid anatomy we can say nothing.

In the third case, we think that the affection is probably purely spinal, but is not so distinctly chorea, rather a choreic form of spasm.

It will be observed that we have been careful not to assert our belief in more than the probability of the spinal cord having been concerned in the production of the peculiar spasmodic movements described. We have wished most of all to call attention to the cord as possibly the parent of certain forms of choreoid disturbances like those seen in the familiar canine chorea so well studied by Horatio Wood and others.

A CASE OF PSYCHIC PARESIS, WITH AUTOPSY.

The following is related by Dr. Inglis in the "Proceedings of the Detroit Medical and Library Association," 1889: An adult male, with unknown history, was seen in a state of mental torpor and apparent right-sided paresis. He appeared to understand questions, but could only bring his mind to bear on any subject a moment. After protruding the tongue he would fail to withdraw it, and in shaking hands would forget to let go. His answers were appropriate, but monosyllabic. The paresis was not a true one, movements of the right side being performed when specially ordered. At the autopsy an infiltrating tumor was found in the white substance of the left anterior lobe, lying chiefly under the middle frontal convolution. A smaller tumor was also found in a corresponding position on the right side and an infiltrating deposit of the same growth existed on the surface of the corpus callosum. ("Medical Record," March 22, 1890).

A. F.

CONTRIBUTION TO THE STUDY OF THE TRAUMATIC NEURO-PSYCHOSES.

By G. L. WALTON, M.D.,

Instructor in Diseases of the Nervous System, Harvard University; Physician to the Neurological Department of the Massachusetts General Hospital.

THE pathology and prognosis of the injuries inflicted upon the nervous system by railway collisions and similar accidents, have furnished, perhaps, as much material for discussion, during the past decade, as any other subject in medical science. The diversity of opinion on this subject is peculiarly unfortunate, in that it is not merely one of scientific interest, but one of great practical importance.

It is curious that the first impetus was given to this distinctly neurological subject by a surgeon, and that his views should have been for so long a time adopted, practically unchallenged, as to have become largely incorporated into the text-books of neurology (Erb, Gowers, Ross) as well as surgery.

Under the influence of Erichsen's views, functional and organic injuries were for a long time indiscriminately classed together under the ambiguous and misleading term, spinal concussion, and a common prognosis was given to all, varying, it is true, in its possibilities, from death to recovery, but leaving the student in doubt, whether there were any method of forming even a probable opinion in a given case, as to whether the worst or best result might be expected.

To Page¹ is due the credit of elaborately correcting this inaccuracy, and of sifting out the comparatively rare cases of organic spinal disease, whose sad course and prognosis had been so long allowed to overshadow and include the more common cases in which no demonstrable lesion existed. To the latter class he first applied the term

¹ Injuries to the Spine and Spinal Cord : Herbert W. Page, A.M., M.C., 1883.

traumatic neurasthenia. The difficulty of injuring the spinal cord as long as its bony covering remained intact, was also first pointed out by this observer. Since, and largely on account of, the appearance of Page's work, a decided, though not unanimous, modification of opinion has taken place regarding the pathology of these cases, though there is still considerable variance regarding their prognosis, even among those who have accepted the theory of a functional basis for the majority of the cases.

The advance in our knowledge of the subject has been doubly rapid through being gained not only from medico-legal observers, but also from clinical investigations in hysteria, more especially those of Charcot and his followers, whose studies have thrown light on the more stabile forms of this protean disease, which has removed the objection originally presented, to classing under this head cases with symptoms comparatively fixed in seat and character. Indeed, as is well known, Charcot at first considered all these cases simply hysteria.

Dr. Putnam,² in 1883, after reporting several cases of traumatic hemi-anæsthesia, called attention to the importance of looking for evidences of typical hysteria, in the chronic as well as in the acute stages of so-called spinal concussion. In the same year, in illustration of the general tendency to withdraw the attention from the spinal cord, the writer,³ in a paper on the cerebral origin of symptoms sometimes classed under spinal irritation, proposed the name "railway brain" as more appropriate than "railway spine" to designate the class of traumatic cases under discussion. This name has been quite extensively adopted, though, as Thorburn comments, it was only a step in the right direction.

Among those who early inclined toward the modified views regarding the effects of trauma on the nervous system, may be mentioned Dana,⁴ who, writing in 1883, very

² Recent Investigations into the Pathology of so-called Concussions of the Spine, etc.: Boston Med. and Surg. Jour., Sept. 6, 1883.

³ Spinal Irritation: Probable Cerebral Origin of the Symptoms, etc.: *Ibid.*, Dec. 27, 1883.

⁴ New York Med. Record, Dec. 6, 1884.

appropriately added hypochondria to the two terms already applied, a term widely applicable in view of the morbid introspection and self-study which does so much to increase and perpetuate the subjective symptoms. This writer, while considering spinal concussion a possibility, preferred to regard the majority of cases thus designated as traumatic hysteria, hypochondria, or neurasthenia, associated more or less with symptoms of injury to the vertebral ligaments and muscles, and to the spinal nerves. The prospect for improvement he considered good, but not that for perfect recovery.

Spitzka⁵ had considered that spinal concussion could produce spinal irritation, a disturbance not to be mistaken for myelitis or meningitis, but at the same time one whose severity should not be lightly regarded.

These theories were in direct opposition to the idea advanced by Westphal⁶ of a diffuse sclerosis set up by the jar, perhaps through the mechanism of minute hemorrhages, as well as to the views of Leyden, which favored organic disease as a common sequel concussion. Among the followers of Westphal were numbered originally in Germany Oppenheim and Thomsen, and in America Knapp⁹ and others. The conscientious and thorough work of Oppenheim in this direction became so generally recognized that his later conclusions¹⁰ designating the majority of the cases as neuro-psychoses have had perhaps more influence than the efforts of any other writer since Page in modifying the general opinion.

Knapp,¹¹ in a later article, adopts the classification of Oppenheim, though still adhering to the view that certain of these cases should be classed under the sclerosis of Westphal.

⁵ Am. Jour. of Neurology and Psychiatry, Aug., 1883.

⁶ Einige Fälle von Erkrankung des nervou Systems nach Verletzung auf Eisenbahnen. Charité-Amalen. v. 379, 1878.

⁷ Klinik der Rückenmarkskrankheiten, B. II., Berlin. 1875.

⁸ Berl. klin. Wochenschrift, 1884 : Archiv. f. Psychiatrie. 1885, vol. xvi.

⁹ Boston Med. and Surg. Jour., Oct. 25, 1888.

¹⁰ Berl. klin. Wochenschrift. 1888, No. 9.

¹¹ Boston Med. and Surg. Jour., Dec. 19, 1889.

Thorburn,¹² in his excellent work on the surgery of the spinal cord, states that recent writers are practically unanimous in agreeing that concussion of the spinal cord is at least an extremely rare lesion, though there is still much dispute as to the significance of the nervous symptoms commonly observed after severe physical or physical and psychical shocks. His views of the pathology and prognosis of the disease do not differ materially from those of Page, though he draws the distinction more closely between hysteria and neurasthenia.

In a recent work by Clevenger,¹³ of Chicago, it is proposed to give to these cases the name "Erichsen's disease," in honor of one whose work this author considers, with some modifications, standard. This writer regards the spinal sympathetic system as the starting point of the pathological process, but reverts in general to the spinal cord as the principal seat of disease, encouraging also the view that myelitis, meningitis, meningo-myelitis, locomotor-ataxia, diffuse and lateral sclerosis are not uncommon sequelæ of trauma.

Seguin,¹⁴ in his admirable summary of recent progress in Sajous' Annual of 1889, based on the articles of Oppenheim, Knapp, Strümpell, Bajinsky, Wolffe and Shaw, considers organic injury to the nervous system a rarity as resulting from the forms of trauma under consideration. He lays much stress on suggestion, both hypnotic and non-hypnotic, discusses deliberate malingering, and emphasizes the prominence of subjective, and absence of objective symptoms. He criticises the too common habit of accepting literally the statements of the patient regarding his own sufferings.

Without exhausting the literature of the subject, it may fairly be concluded that there is at present a very general, though not unanimous tendency to abandon the theory of spinal concussion as at all widely applicable to the class of cases under consideration, and to regard the majority of

¹² Contribution to the Surgery of Spinal Cord by Wm. Thorburn, B.Sc., B.Sc., M.D. (Lond.).

¹³ Spinal Concussion, etc., by S. V. Clevenger, M.D. Phila., 1889.

¹⁴ Vol. iii. Q.

the genuine cases as identical with already recognized functional forms of disease, rather than as cases of organic spinal injury.

My own experience has lead me from the first to regard disease of the spinal cord resulting from trauma as of comparative rarity, when no dislocation or fracture has occurred, while Seguin's conclusion regarding the predominance of subjective symptoms, and the degree in which we are generally dependent on the patient's statement, are fully justified by the majority of the cases which have come under my observation.

I have especially noted the rarity of such symptoms as local atrophy, fibrillary twitching, bed-sores, cystitis, disturbance of electrical reactions, involuntary rigidity, spastic gait, toe-drop, ankle clonus, pupil irregularities, and disordered reflex (beyond mere activity, which, as Seguin states, is alone of no diagnostic value). The symptoms nystagmus, and incontinence of urine, which Knapp inserts in his *résumé*, I have found notably absent. A number of the patients have *complained*, it is true, of retention or incontinence, but absence of palpable proof of these symptoms has forced me in every instance of the class of cases under consideration, to regard the statement with considerable skepticism. In fact, questioning the patient alone has generally sifted down the complaint to frequency or delay of micturition, symptoms which are common in functional cases, non-traumatic, as well as traumatic, and which, as compared to the true retention which causes overflow unless the catheter is used or to the true incontinence resulting from organic disease, with its attending annoyances both to the patient and his friends may almost be regarded as trivial. Nor do even these mild forms of bladder trouble necessarily exist in every case where claimed, but they must be reckoned among those symptoms for which we have no evidence beyond the patient's statement. In the same category, I should agree with Seguin in placing anæsthesia, both general and special, and of course, also, hyperæsthesia, and paræsthesia, as well as pain. I have become so convinced that in the case

of a practised simulant the various tests for anæsthesia, even including the wire brush, are non-conclusive, that in many cases I make no attempt to either establish or confute the claim, but set it down as a symptom for which I have the patient's statement only, just as for the pain, sensitiveness, bad dreams, and other symptoms; no one but himself knowing whether it is present or not, and in view of Seguin's observations regarding suggestion, it may, perhaps, in genuine cases, sometimes even be doubtful whether the patient himself has any very definite consciousness of the exact severity of these subjective symptoms.

In a similar category, I should place certain symptoms occasionally seen, upon which Knapp has laid stress as almost of necessity pointing to organic disease; such are: the complaint of a constriction about the waist, swaying with closed eyes, tremor, and a staggering gait. The sense of constriction may be as easily complained of by the hysterical or neurotic patient or the simulant as impotence or any other subjective symptom, while swaying, tremor, and staggering may occur as symptoms of functional trouble or general debility, or again may be easily simulated. A tremor of the head, for example, may be kept up a considerable time without fatigue, by resting the hands on a walking-stick upright between the knees, or upon the edge of a table. A rythmical movement may be started by the arms in this position by which the head is caused to oscillate forwards and backwards, the movement being a comparatively natural one, and causing little fatigue. This variety of tremor, as well as less successful attempts at tremor of single extremities, I have seen, where I was convinced of the presence of simulation. I have also seen a voluntary tremor in the leg which was apparently an attempt to produce an ankle clonus.

It will be noted in the *résumé* of cases following, that a large number complained of loss of power. The loss was, as a rule, quite generalized, involving one or more entire extremities. In some of these cases there was palpable fraud, shown, for example, by the patient making with ease, when his attention was diverted, the same movements

which he had declared himself absolutely unable to perform. In others the loss appeared genuine, while in others it was impossible to decide to a certainty whether the case was one of fraud or hysteria. In none of the cases which I have classed under the neuro-psychoses was the gait characteristic of organic disease, the foot being generally everted and dragged with its whole length scraping the floor, or again, the foot would sometimes clear the ground entirely, sometimes scrape with the heel as well as the toe, a gait which, if genuine, points rather to hysteria than to organic disease. In some cases oddities of gait appeared which could not be brought under any recognized class of paralytic gait.

Typical hysterical contracture was present, I think, in only one case.

Allied to the active reflex I have often found a rapid, but otherwise normal pulse, a symptom upon which I have seen great stress laid in courts, but which has generally been either the result of excitement or of weakness, or of a neurotic or hysterical tendency, associated, for example, with large pupils. In investigating this symptom, I have been surprised to find how easily the normal pulse is elevated to 100 and over, even on moderate excitement. Indeed, a pulse-beat of 90 to 100 is by no means an extreme rarity in a healthy individual, whether male or female (though, of course, more common in the female), considerations which should make us chary of attributing too much importance to a rapid but otherwise healthy pulse. The fact that a rapid pulse is often found in locomotor ataxia is hardly sufficient ground for advancing this symptom as proof of organic disease of the cord in the absence of other objective signs. The same is true of the active reflex, unaccompanied by clonus, especially when it is accompanied by the complaint of disagreeable sensation up or down the leg, into the groin, back or head, or when accompanied by the peculiar give, or jump, which neurasthenic and hysterical patients sometimes exhibit. It would certainly seem that these symptoms, occurring, as they generally have in my cases, in connection with such sub-

jective symptoms as inability to confine the attention, emotional tendency, flushes and chills, or nervous irritability, rather than with definite objective signs, should cause us to look upon them, where considered symptoms at all, as symptoms of a condition known to be present, rather than of one for which we have no further evidence.

My experience has lead me to coincide with Page's view, as already stated, that it is extremely difficult to injure the spinal cord excepting through the medium of its bony canal. I do not mean that such injury is impossible, in fact, during the period in which the cases referred to in this paper have come under my observation, I have seen two cases (elsewhere reported)¹⁵ of undoubted hemorrhage into the substance of the spinal cord, each with atrophic paralysis with degeneration reaction of arm muscles on one side, and one with spastic condition of the corresponding leg, the symptoms appearing directly upon the accident, which was in each case a severe one. I have also seen a case in which the symptoms, including spastic gait and ankle clonus pointed to hemorrhage about the cord, and several of injury to nerve roots, with characteristic distribution of motor and sensory paralysis, as investigated physiologically by Ferrier and Yeo,¹⁶ anatomically by Herrigham,¹⁷ and clinically by Ross,¹⁸ Thoburn¹⁹ and others. In all these cases objective signs of spinal injury were present, and only in such cases have I felt justified in making the diagnosis of organic injury to the spinal cord which did not implicate the bony canal, the *possibility* that minute hemorrhages may take place in an organ hardly seeming to me sufficient ground for making such a diagnosis during life, unless certain symptoms have been established as diagnostic of that condition. As regards the term spinal concussion, I have not yet seen a case in which I felt justified in making this diagnosis; I do not think it is to be found in the books of

¹⁵ Cases of Functional and Organic Injury, etc.: Boston Med. and Surg. Jour., Feb. 4, 1886.

¹⁶ Proceedings of the Royal Society, No. ccxii., p. 12: Brain, 1882.

¹⁷ *Ibid.*, No. ccxliii., 1886, p. 255.

¹⁸ Brain, Jan., 1888.

¹⁹ A Contribution to the Surgery of the Spinal Cord.

the neurological department of the Massachusetts General Hospital.

There are one or two points regarding the protection of the spinal cord which have occurred to me, beyond those to which attention has already been called. The fact has been emphasized that the cord is an organ comparatively light in weight, that it is suspended in a fluid comparatively greater in quantity than that surrounding the brain, and that it is further surrounded by membranes, areolar adipose tissue, and a thick plexus of blood-vessels. I would also call attention to the fact that the construction of the vertebræ is such that the force of a blow upon the spinous process must pass *around* the spinal cord, to be principally expended upon the bodies of the vertebræ, perhaps also in part to the ribs. Again, besides the flexibility of the vertebral column as a whole, which tends to lessen the force of a blow, the downward inclination of the spinous processes in the greater part of the column would tend to tilt the corresponding vertebra, thus still further adding to the elasticity of the protecting structure.

As regards locomotor ataxia, lateral or disseminated sclerosis, and similar diseases, it seems to me that our knowledge of the pathology and course of these diseases, together with their insidious mode of onset, should make us extremely chary of accepting a traumatic origin, even though isolated cases have been reported by most eminent authority, as dating from an accident. Some allowance must be made for coincidence. I have recently seen, for example, a patient with typical locomotor ataxia, who, with no claim for compensation, dates his trouble from a strain of the back; the strain proving, however, on investigating the case, to be nothing more than a coincidence, an acknowledged specific history pointing to the more probable etiology.

Such cases might be multiplied indefinitely, and should lead us to avoid the tendency in examining legal cases, to accept at once statements regarding previous health, by patients who do not always recognize their own condition, thus neglecting, in cases involving large sums of money, to

use the caution we should apply to outside cases, merely as a matter of scientific interest, or for ordinary purposes of accuracy.

Analysis of one hundred successive cases as found in my notes, where nervous symptoms were complained of, and where the question of damages had arisen or was likely to arise, gives the following results :

There were two cases of vertebral fracture, one of vertebral dislocation, one of injury to cervical nerve roots, two of probable neuritis, one of long-standing spinal sclerosis, one of old infantile paralysis, one of extensive atheroma, one of choroiditis, four of heart disease, one of cystitis of local origin, and two of severe constitutional disease with emaciation (phthisis, scrofula). Four of these seventeen cases have died, one directly from the effects of broken back followed by bed-sores and cystitis, and one from phthisis; in the other two, designated above as neuritis and atheroma, the exact nature of death has not been ascertained. In one of the remaining cases I am still in doubt as to the diagnosis. This was the case of a brakeman who was thrown from the top of a freight-car, striking on his back. When I saw him, the back was held stiffly and the legs rigid. He appeared able to get about only with great difficulty and a short distance by the aid of crutches. He complained of loss of sensation as well as power in both legs. Satisfactory examination was impossible on account of the patient's unwillingness to be examined. No irregularity of the back was found. There was present, however, not only an active knee-jerk, but a marked and continuous ankle clonus on both sides, and on the strength of this symptom I reported to the road by whom I was sent to examine him that, notwithstanding the incompleteness of the examination, I was satisfied that there must be organic injury to the spinal cord. Upon this report the case was settled for a large sum. Not long after I learned that he had recovered after a course of treatment at some resort, and at present I am informed that he is serving a term in the State prison, having been convicted of a rape committed shortly after the settlement. Whether

this was a case of hemorrhage about the cord with absorption, or whether it was an exceptional case of functional disorder or of simulation, in which ankle clonus was present, or whether some other explanation can be offered, this exceptional history would not of course alter the fact that, as a rule, a marked and continuous ankle clonus denotes organic injury or disease. That the symptom was simulated I can hardly believe.

The remaining eighty-two cases, with the exception of the simulants, would fairly come under the class designated by Oppenheim as neuro-psychoses, either alone or complicating surgical injuries. (These surgical injuries, in some cases serious, will be left out of consideration in this paper as foreign to the subject.)

In a large number of the cases there was gross exaggeration, and in a certain number deliberate fraud was detected; but for the purpose of this analysis, which has rather to do with the question of organic as against functional trouble, it will not be necessary to separate these classes. In a large number of these cases the claim of spinal injury was made, either without specifying the pathology of the injury, or as concussion, sclerosis, myelitis or meningitis, an opinion in which I could in no case concur.

The nature of the trauma ranged from slight jars and moderate blows on various parts of the body to violent collisions and severe falls. The nervous symptoms bore, on the whole, comparatively little relation in point of severity to the violence or direction of the trauma after the first effects were over. Identical symptoms were complained of, for example, after blows upon the extremities, with those following blows upon the back and head, or injuries causing a jar of the whole body, which shows at least that a concussion of the spine or even of the brain, is not an essential factor in their production.

The time of my examination ranged from one week to three and one-half years after the accident.

Pain in the head and back was complained of in forty-six cases; in the back alone, in twenty-four; in the head

alone, in six, making a total of seventy-six cases complaining of back, in the head or back, or both. In a lesser number pains, without physical signs, were complained of in the abdomen, sides, arms, legs, neck, chest, groin, and "all over."

Sensitiveness was noted in the back in forty cases, and in a lesser number in various other regions, including the head, chest, sides, abdomen, and extremities. It was common for sensitiveness and pain to be complained of in the exact region where blows had occurred, at intervals of months and even of years, after the accident, after all external signs had passed away. This is a symptom difficult to trace to spinal disease, but readily understood when the mental element is considered, peculiar to hysteria and hypochondria, even without having recourse to the theory of simulation, which was of course, in some cases, present. Headache and backache, however, seemed to occur quite irrespective of the nature or seat of injury.

Following are the other symptoms complained of as far as found in my notes:

Blurred vision, vertigo, menstrual irregularities, deafness, constipation, diarrhœa (one only), confusion, nervousness and irritability, loss of memory, loss of sleep, general weakness, nausea with or without vomiting, frequent micturition, loss of sexual power, poor appetite, cold extremities, palpitation, indigestion, bad dreams, cramps, tinnitus aurium, trembling, weakness in the back apart from general weakness, inability to read, inability to confine the mind to work, photophobia, emotional tendency, muscular twitching, paræsthesia, faintness, sensation of heat, flushes and chills, despondency, *muscæ volitantes*, hallucinations of sight, inability to confine the attention, change in disposition, difficult micturition, hysterical attacks, sense of swelling in the side, indescribable sensation, dragging in the abdomen, night-sweats, sense of fear, temporary diplopia, temporary ptosis, drawing in the neck, leucorrhœa, increased perspiration, apathy, light feeling, difficulty in swallowing, difficulty in talking, a sense of pulling in the stomach, sense of constriction, sense of swelling in the eyelids, fluttering of

the stomach, sense of impending ill, stupid feeling, sense of pulling in the eyes, of pulling in the head, of pressure in the head, of tightness about the hip, of an electric shock, of the heart being squeezed, of the head separating, sensation as if knocked upwards, epistaxis, increased stuttering.

Forty complained of numbness: ten of these complained of more or less well-defined left hemi-anæsthesia, seven of right hemi-anæsthesia, ten of numbness in the lower extremities, two in the upper, two in the trunk, and four of general numbness. The anæsthesia was in no case limited to any special nerve-region, but was generally distributed—bounded, for example, by a line around the limbs, or by the median line of the body. The numbness remained in most cases fixed in character and situation, in some it varied in seat, in others was said to be intermittent.

Thirty complained of specialized loss of power, seven in the left arm and leg, four in the right arm and leg, twelve in both legs, eight in the left leg, one in the right leg, one in both arms. In three of these cases there was staggering gait, and in a number the gait was slow and stiff, but in most the character was that previously described. The loss of power was in no case limited to certain muscles or groups of muscles, but generally included whole extremities.

In none of these eighty-two cases was local atrophy of any muscle or group of muscles found, though general lack of tone of the muscular system was found in quite a number, evidenced by flaccidity. The general nutrition, including that of the muscular system, was good in sixty, fair in eighteen, poor in three, not noted in one.

The tendon reflex was in most cases within the average of health. In thirty-one it was noted as very active (unaccompanied by clonus), and in a considerable number of these the activity was accompanied by a disagreeable sensation. In no case was ankle-clonus present.

A word with regard to the tendon reflex: This phenomenon varies in health from practically wanting to quite violent, the former condition occurring only in isolated cases, the latter less infrequently. I have in mind an instance of a perfectly healthy person in whom repeated trials

by neurologists have failed to produce a trace of reflex, even with the aid of re-enforcement.

The fact that these cases occur, however rarely, prevents our making a diagnosis of organic disease upon this symptom alone, a fact not always recognized. Again, a lively reflex is not infrequently mistaken in these cases for the exaggerated reflex of organic disease of the motor tract, while in reality pointing either to nothing abnormal or to a functional disturbance.

The pupils in general were alike and reacted normally to light. In four they were unlike (the left being in each case the larger), but both reacted perfectly. In one case seen recently, a pupil-irregularity was present which does not come under these heads; but, as the case is not yet settled, I am not at liberty to discuss it at present.

Disturbance of electrical reaction was found in none; this test was made in most of the cases where any complaint of loss of power was made as well as in many others.

The legs were measured in all cases where complaint was made of this part of the body, and in many others. In the fifty-seven cases noted they were alike in twenty; the right was larger in twenty-one, the left in sixteen, the measurements being taken at the calf. In those cases in which the right was larger, the difference was one-sixteenth inch in one, one-eighth inch in eight, one-fourth inch in eight, three-eighths inch in three, five-eighths inch in one. In those in which the left was larger, the difference was one-sixteenth inch in two, one-eighth inch in four, three-sixteenths inch in one, one-fourth inch in seven, three-eighths inch in one, one-half inch in one. No greater differences than these were found. It is evident that these measurements varied little, if at all, from those of health, as shown, for example, by series of measurements made by Dr. M. H. Richardson. Nor did the difference always correspond to the leg complained of, nor, in fact, were the legs complained of in all the cases where difference was noted. These measurements were in striking contrast to that of the case mentioned above as infantile paralysis, in which the difference at the calf was three inches. In regard to meas-

urement of the calves, I have found it more accurate to measure the largest part for comparison, making, if necessary, a series of measurements, and noting the largest on each leg, than to first measure down or up from a fixed point. In measuring the thigh, however, the latter plan is of course necessary.

The comparison of measurements taken at any one time with those taken at another time is fallacious where the difference is no greater than those recorded here, inasmuch as the tape may be drawn more loosely at one time than another. This can be controlled more accurately, however, where it is a question of measuring the two legs at one time. Comparison of the measurement of two different physicians is even more fallacious.

Stiffness of the back was noted in a considerable number of the cases. In two cases old Pott's disease was present.

The ages of these eighty-two cases ranged from fifteen to seventy-five. With the exception of one case at fifteen, I have never seen these symptoms complained of under seventeen, though suits are not infrequently brought for other forms of injury in children. This is a peculiarity of the neuro-psychoses worthy of consideration.

The character of the pulse was generally normal. Its frequency was noted in fifty-nine cases, thirty-one male and twenty-eight female.

Of the male pulses, one was 68, eight were between 70 and 80, nine were between 80 and 90, three were between 90 and 100, two were between 100 and 110, two were between 110 and 120, four were between 120 and 130; the average was $90\frac{1}{2}$.

Of the female pulses, one was 68, two were between 70 and 80, six were between 80 and 90, seven between 90 and 100, six between 100 and 110, two between 110 and 120, two between 120 and 130, and one was 135; the average was $95\frac{3}{4}$.

This point I have already alluded to. It is difficult to eliminate the factor of excitement under examination in these medico-legal cases, for a great degree of excitement is not necessary to cause marked acceleration of the pulse.

I have a number of times found pulses over 100 in the case of healthy professional gentlemen just about to go upon the witness-stand, none of whom showed any external sign of excitement; in one such case the pulse was 120. In corroboration of these statements, Dr. Prince and Dr. Ham inform me that their records as pension-examiners show so continuously high a pulse-rate, whatever the nature of the complaint, that they have become accustomed to rely entirely on the character without reference to the rate. Dr. Carter has given me a list of pulses from neurasthenic patients at the clinic, which ranged all the way from 80 to 120.

The objection may be made to my reports that another observer might have discovered loss of electrical reaction or other sign of organic disease, where I did not. In fact, such symptoms have been claimed in many of these very cases; in no case, however, I believe, by a neurologist. Nothing is more common than for the general practitioner to be deceived on this point, through lack of familiarity with the motor points, and the strength of current required to produce contractions, and the allowance to be made for cutaneous resistance. I have not infrequently seen the patient jump with the presumably well leg and fail to do so with the other, when the electricity was applied, either because of anæsthesia of the latter limb or through fraud; this jump I have seen mistaken for an electrical reaction. Again, I have heard the remarkable statement made that the motor points must have been stimulated, as the electrodes were applied all over the extremities.

Page has been criticised as having laid too much stress upon the strain of the back in explaining many of the symptoms referred to that region. As already stated, I have noted rigidity of the back in a considerable number of the cases, and made the diagnosis of strain, either of muscles, ligaments, or both.

In a large number, however, the back was perfectly flexible, as shown by bending forward and backward, sideways, and twisting. That this symptom is sometimes simulated has been shown by certain patients who have

declared themselves unable to make these movements, but who have exhibited perfect flexibility of the column while removing their underclothing. Where severe wrench of the back is present the injury is by no means to be regarded as trivial, this affection being sometimes extremely persistent and troublesome, although its tendency is, in general, in the direction of improvement. As Thorburn has pointed out, the rapidity of improvement in all the symptoms will depend largely upon the surroundings of the patient, as well as upon his temperament and previous condition. Inasmuch as the active exercise of will-power is bound to be an element in the improvement, it is not surprising that this improvement is frequently postponed until after the depressing surroundings and uncertainties incident to legal proceedings have been removed, together with the inducements to morbid introspection thereby afforded.

This being true, even in cases where no deception is present, it is of course more so where the patient willingly drifts into invalidism. Deliberate simulants are quite apt to appear worse and worse as the trial approaches.

My experience, as far as I have had opportunity to follow cases where no claim for damages existed, or to follow legal cases after such claim has been adjusted, does not justify the assertion that a common tendency is downward, although I have not infrequently seen cases appearing worse and worse up to the time of trial (sometimes especially so within a few days of the trial). I am convinced that the case where this downward tendency continues afterward is a rarity among the class under consideration. In fact, even in organic injuries, as fracture of the vertebræ, this gradual downward tendency is hardly the rule, inasmuch as, in most such cases, death either follows within a comparatively short period or improvement sets in up to a certain point, to be followed by standstill. As an exception to this rule may be mentioned Bernhardt's²⁰ case of gradually developing spinal tumor discovered by post-mortem examination. With regard to the case of general paralysis, described by the same author,

²⁰ Deutsche med. Wochenschr., 1888, No. 13, p. 245.

the connection between the accident in 1875 (from which the patient recovered, so far as to be able to do active work satisfactorily), and a typical general paralysis, disclosing itself in 1886, is not absolutely established.

In conclusion, such careful clinical work as that of Page should not be undervalued on the ground of necessary bias. On the contrary, such efforts should rather tend to further the cause of justice in the long run, to plaintiffs as well as to defendants, for if we regard functional (to say nothing of fraudulent) cases as sufferers from organic disease of the spinal cord, the tendency will be, as the recovery of more and more of these cases goes on record, to produce distrust, not only of medical acumen, but of the statements of claimants in general, so great as to deprive of their just dues cases of hopeless organic disease, even when vouched for by most distinguished medical authority.

CEREBRAL COMPLICATIONS OF EAR DISEASE.

The "Medical Age," Jan. 10, 1890, quoting from the "Deutsche Med. Zeitung," gives Dr Otto Körner, of Frankfurt, as authority for the statement that all cerebral complications of ear disease are more frequent on the right side than on the left. Of thirty-one cases of cerebral abscess, eighteen occurred on the right side from disease of the ear, and twelve on the left, while in one there was abscess on both sides following ear trouble. Of sixty-one cases of thrombosis of the lateral sinus, thirty-five were on the right and twenty-six on the left side. Out of twenty-three cases of meningitis from ear disease, seventeen were on the right side and three on the left. This makes a total of one hundred and fifteen cases, of which seventy showed abnormalities on the right side, forty-four on the left, and one bilateral. Körner traces the greater frequency of right-sided troubles to the fact that the groove for the lateral sinus passes further forwards and outward into the petrous and mastoid bones on the right than on the left side. Therefore the bony wall separating the sinus from the mastoid antrum is thinner on this side. Add to this the fact that in brachycephalic skulls the anterior cerebral fossa is considerably nearer the mastoid than in the dolichocephalic, and it will be perceived why cerebral complications more readily ensue in brachycephalic skulls.

A CASE OF LOCOMOTOR ATAXIA ASSOCIATED WITH NUCLEAR CRANIAL NERVE PALSIES AND WITH MUSCULAR ATROPHIES.¹

By FREDERICK PETERSON, M.D.,

Attending Physician to the New York Hospital for Nervous Diseases.

THE case whose clinical history I am about to report has been a patient of mine at the New York Hospital for Nervous Diseases on Blackwell's Island since March 27, 1890. After having made a careful study of his condition, I discovered that he had been under the charge of Dr. E. C. Seguin at the Manhattan Eye and Ear Hospital from 1884 to 1886, and that Dr. Seguin had described the features of the case as they then existed in the JOURNAL OF NERVOUS AND MENTAL DISEASE for May, 1888. It is the first of five cases of ophthalmoplegia reported by the author in that journal.

There have been so many new developments in his condition during the past four years that I may be pardoned for briefly outlining his complete history from the first observations made to the time when he became a patient of my own.

M. J. T., now thirty-seven years of age, had a chancre and secondary symptoms fifteen years ago, that is, about 1875.

In 1882, he discovered, one morning, dimness of vision and external strabismus of the left eye with diplopia. A little later he had shooting-pains in the legs, occasionally in the arms.

In 1883, he had a momentary loss of consciousness, and fell, cutting his head. His left testicle also became swollen and hard during this year, and he was on specific treatment at Hot Springs for some time.

¹ Read before the American Neurological Association at Philadelphia, June 4, 1890.

In 1884, he had partial double ptosis. In the right eye, the internal rectus, inferior oblique, and sphincter iridis were paralyzed, and the superior and inferior recti feeble. The other muscles were normal. In the left eye the muscles supplied by the third nerve acted variably and feebly. The other muscles were normal. Both pupils were completely motionless to light and accommodation, the left larger than the right. There was overaction of the occipito-frontalis. Ophthalmological examination resulted as follows:

$$\text{Right V. } \frac{20}{70} - \frac{1}{18} = \frac{20}{40}.$$

$$\text{Left V. } \frac{20}{30} - \frac{1}{18} = \frac{20}{40}.$$

Accommodation right $\frac{1}{10}$, left $\frac{1}{14}$. No lesion of the optic nerves.

The left cheek was a little inactive, and there was a mild paresis of the right hand. (Dynamometer: R. 42—44, L. 45.) No Romberg symptom. Knee-jerks exaggerated. Both feeble and involuntary micturition.

In 1885, the ptosis was nearly total on the left, but partial on the right side. In the right eye the condition of the muscles was unchanged, while in the left they had improved so much that they acted almost normally. Some paresis and atrophy of both temporal and both masseter muscles were now noted.

In 1886, when lost sight of by Dr. Seguin, the ptosis was a little greater, the bladder still parietic, and the masticatory muscles unchanged. No marked facial paresis. The knee-jerks previously exaggerated had fallen to about normal.

Dr. Seguin, writing in 1888, said of this case:

"The fulgurating pains, with hyperalgesia, and the fall in degree of knee-jerk, during two years of observation [1884-1886], would seem to justify a suspicion of incipient posterior spinal sclerosis."

Since 1886 until the present time there has been gradual progress in his disease. The condition of his eyes is now as follows:

In the right all of the muscles external and internal, except the rectus externus, are completely paralyzed. The rectus externus is parietic, and when moved exhibits clonic spasm. In the left eye there is almost complete ophthalmoplegia externa et interna, but the superior and inferior

oblique muscles still move a very little and very feebly. Both pupils are widely dilated, equal and immobile. Divergent squint of right eye, pulled outward by the paretic rectus. Accommodation paralyzed. Vision is unchanged.

As far, therefore, as the innervation of the ocular musculature is concerned, we have now lesions affecting both third nerves, both fourth nerves and both sixth nerves.



The weakness and atrophy of the masseter and temporal muscles are, if anything, more pronounced than before. In eating he has to support and assist the lower jaw with his hands. The two pterygoids on each side are also paretic. He cannot move the jaw forward or from side to side. The temporals and masseters do not react to faradism. The

wasting is evident in the photograph. There is no anæsthesia of the face. The left side of the face is a little paretic. It will be noticed in the photograph that the action of the occipito-frontalis is stronger and the nasolabial fold deeper on the right than on the left side. The tongue deviates slightly toward the left. The other cranial nerves are normal.

The electric reactions in the face and tongue muscles are normal.

There is still evidence of weakness in the right hand, for the dynamometer in three trials registers R 40-30-30, L. 35-35-38.

He has now well-marked *tabes dorsalis*. There is great ataxia of all four extremities. He cannot walk without assistance. The knee-jerks have disappeared altogether. There is numbness, anæsthesia and analgesia in all of his fingers. He cannot pick up a pin.

In both legs as far as the knees there is almost complete tactile anæsthesia. Muscular sense is entirely lost in both feet. There is analgesia and diminished tactile sensibility over the whole of the lower extremities and on the trunk as far as the umbilicus. There is almost total anæsthesia to heat and cold from the feet to the hips, and deferred sensation is a constant, and *allocheiria* an occasional phenomenon. The plantar and cremasteric reflexes fail.

He has incontinence of urine and the anal sphincter is weak. He has never had any crises. He has now no lightning-pain and no girdle sensation.

But in addition to the fully-developed locomotor ataxia and the nuclear paralyses mentioned, the patient now presents also some very interesting trophic disturbances. I will not include a rather remarkable fracture of the left clavicle at the junction of the inner and middle thirds (the outer portion is dislocated downwards one inch), for it is not apparently of recent date, and the patient never knew of its existence until I discovered it, and its history is therefore obscure.

Certain muscular atrophies are, however, very prominent. Besides that of the muscles supplied by the motor

branches of the two trigemini already described, there is conspicuous wasting of the right trapezius with a degenerative reaction.

In the left upper extremity there is complete paralysis of the two long extensors of the phalanges of the thumb, and atrophy and paresis of the abductor minimi digiti and of all of the interossei and lumbricales, with degenerative reaction. The left hand assumes almost the position of a *main en griffe* when at rest. The illustration of the two hands reveals the deformity only to a slight extent.

On the right side there is almost complete atrophy of the opponens and abductor pollicis.

Considerable wasting is apparent in the adductors of both thighs, more upon the right side; and there is a marked difference in the circumferences of both the thighs and the legs, as is evident from the following measurements:

<i>Circumference of</i>	<i>Right</i>	<i>Left.</i>
Thigh, 13 cm. above patella.....	49 -37	42.5-39
Leg, 13 cm. below patella.....	29 5-28	27.5-26

From this it is clear that the right thigh is smaller than the left, whereas the left leg is smaller than the right by some two cm.

The electrical reactions are quite normal in all of the muscles of the lower extremities, except the adductors of the thigh and the sartorius of the right side, where the contractions to faradism are weak and "wabbling."

Most of the muscles of the body were examined with both the faradic and galvanic currents, and those in which partial or complete degenerative reaction was present are exhibited in the accompanying table.

ELECTRICAL REACTIONS IN ATROPHIED MUSCLES—CASE OF M. J. T.

<i>Muscles.</i>		<i>Faradic (Primary).</i>	<i>Cm.</i>	<i>Galvanic.</i>	<i>No. of Grenet cells.</i>
Left.	Ext. primi internodii pollicis.	No contraction.....	12	No contraction.	31
	" secundi "	" "	12	" "	31
	First interosseus.....	Slow "	10	Slight K C C.....	31
	Second "	No "	12	No contraction.	31
	Third "	" "	12	An C C > K C C..	31
	Fourth "	Slow, wabbling contraction..	12	K C C > An C C..	18
Right.	Abductor minimi digiti.....	Slow contraction	12	K C C > An C C..	27
	Trapezius.....	Contraction in parts... ..	12	A C C = K C C....	16
	Abductor pollicis.....	No contraction.....	12	A C C > K C C....	31
	Opponens pollicis.....	Slow, wabbling contraction..	12	A C C > K C C....	31
	Sartorius.....	" " "	12	A C C > K C C....	25
	Adductors of thigh.....	" " "	12	A C C = K C C....	25

Summary.—The main features of the case, then, may be summarized as follows: The patient has a number of bilateral motor-cranial nerve palsies, viz.: the third, fourth, fifth and sixth nerves; and for convenience of historical survey and clearness I have made use of Dr. Starr's diagram of the third nerve nuclei,² adding thereto very schematically the nuclei of the other affected nerves, and inserting the dates of their lesions, as pointed out in the clinical history.

SCHEMATIC REPRESENTATION OF NUCLEI OF NERVES AFFECTED
TO SHOW THE HISTORY MERELY.

Left.	Median line.		Right.	
<i>Sphinct. iridis.</i> Paralyzed, 1884.	<i>Ciliary musc.</i> Paralyzed, 1890.	III.	<i>Ciliary musc.</i> Paralyzed, 1890.	<i>Sphinct. iridis.</i> Paralyzed, 1884.
<i>Levat. palp.</i> Feeble, 1884.	<i>Rect. int.</i> Feeble, 1882 to 1884. Normal, 1885. Paralyzed, 1890.		<i>Rect. int.</i> Paralyzed, 1884.	<i>Levat. palp.</i> Feeble, 1884. Paralyzed, 1885
<i>Rect. sup.</i> Feeble, 1884. Normal, 1885. Paralyzed, 1890.	<i>Rect. inf.</i> Feeble, 1884. Normal, 1885. Paralyzed, 1890.		<i>Rect. inf.</i> Feeble, 1884. Paralyzed, 1890.	<i>Rect. sup.</i> Feeble, 1884. Paralyzed, 1890.
<i>Obliq. inf.</i> Feeble, 1884. Normal, 1885. Almost completely paralyzed, 1890.				<i>Obliq. inf.</i> Paralyzed, 1884.
	<i>Obliq. sup.</i> Almost completely paralyzed, 1890.	IV.	<i>Obliq. sup.</i> Paralyzed, 1890.	
<i>Temp.</i> Feeble, 1885. Feeble, 1890.	<i>Masseter.</i> Feeble, 1885. Feeble, 1890.	V.	<i>Masseter.</i> Feeble, 1885. Feeble, 1890.	<i>Temp.</i> Feeble, 1885. Feeble, 1890.
<i>Pteryg. ext.</i> Feeble, 1890.	<i>Pteryg. int.</i> Feeble, 1890.		<i>Pteryg. int.</i> Feeble, 1890.	<i>Pteryg. ext.</i> Feeble, 1890.
	<i>Rect. ext.</i> Normal, 1884. Normal, 1885. Paralyzed, 1890.	VI.	<i>Rect. ext.</i> Normal, 1884. Normal, 1885. Feeble, 1890.	

The patient has also exhibited slight traces of a crossed paralysis for four or more years. He has, furthermore, locomotor ataxia, as shown by the occurrence at one time

² Ophthalmoplegia Externa Partialis. By M. Allen Starr, M.D., Journal of Nervous and Mental Disease, May, 1888.

of lightning-pains and by the presence now of ataxia, widely-distributed anæsthesias, failure of knee-jerks, and ocular, vesical and anal symptoms.

Finally, he presents marked trophic changes in numerous muscles.

Pathology.—As to the morbid processes which underlie these various manifestations, there is, in the first place, undoubtedly a sclerosis of the posterior columns of the spinal cord.

The ophthalmoplegia is, of course, nuclear. Read in one way the symptoms on the side of the cranial nerves, taken in conjunction with the muscular atrophies and paralyses elsewhere, certainly very closely resemble the syndrome so well described by Dr. Sachs in his paper before this Association last year, under the title of Polioencephalitis Superior and Poliomyelitis.³ There is no reason to suppose that polioencephalitis superior and chronic poliomyelitis could not occur in combination with a posterior sclerosis, and it is by no means certain that the case described by Dr. Sachs may not ultimately develop locomotor ataxia, although the absent knee-jerks in both legs and the vesical and anal symptoms in his case may be explained on other grounds. On the other hand, however, these nuclear palsies, more especially of the motor nerves of the eye, are so common in tabes dorsalis, and the investigations of Déjerine,⁴ Nonne⁵ and others have demonstrated that muscular atrophy is not infrequently associated with locomotor ataxia. Déjerine reports 11 cases in 106, and in five of these he made a histological examination, finding the anterior horns normal, but a degenerative neuritis in the affected peripheral nerves. The most important matter to be settled in this case is whether the muscular atrophies are due to peripheral or central lesions. Speculation upon the question would seem to be of very little utility, and its solution must be left to the hoped-for autopsy.

³ Amer. Jour. Med. Sciences, Sept., 1889.

⁴ Gazette méd. de Paris, March 10, 1888, and paper before Société de Biologie, 1889.

⁵ Arch. f. Psych., vol. xix.

It has been assumed by a number of authors that total paralysis of all the muscles supplied by the third nerve implies not a nuclear, but a nerve-trunk palsy. Thus Dr. Starr⁶ says: "If all the muscles of the eyeball supplied by the third nerve are affected, *including the iris*, the case is one of total peripheral paralysis of the third nerve, and the lesion lies on the base of the brain, and may in time implicate other cranial nerves."

In my case all the muscles of both third nerves are totally paralyzed, including both irides, and yet there is every reason to believe that the palsies are nuclear. It would at least be difficult to conceive of a lesion at the base of the brain so widely and so symmetrically distributed as to affect the trunks of both third nerves, both fourth nerves, both sixth nerves, and the motor portions of both trigemini, yet permitting the escape of the sensory portions of the latter.

My own diagnosis of the disease from which the patient suffers is indicated by the title of this paper.

PSEUDO-DISSEMINATED SCLEROSIS FOLLOWING VARIOLA.

("Montpellier Médical," January 1st, 1890). The patient, thirty-four, after a mild attack of variola, manifested nervous symptoms: constant trembling of the head, intention-tremor in the limbs, nystagmus, and difficulty of speech. In three months, under solanine, the greater part of these troubles disappeared. The condition bore a decided resemblance to multiple sclerosis. Was it, however, dependent on any structural change in the nervous system? There are records of cases (Westphall, Babinski, Malguire) that present outward and visible signs of multiple sclerosis which lack confirmation at the autopsy. The rapid course and sudden termination of the disorder suggest its analogy to such functional disturbances.

⁶ *Loc. cit.*

A CONTRIBUTION TO THE PATHOLOGY OF THE LARYNGEAL AND OTHER CRISES IN TABES DORSALIS.*

By IRA VAN GIESON, M. D.,

First Assistant at the Laboratory of the Alumni Association of the College of Physicians
and Surgeons, N. Y.

FOR the opportunity of presenting this case of tabes, in which laryngeal crises, one of them proving fatal, are associated with a chronic neuritis of the roots of the vagus and accessory portion of the eleventh nerves on both sides, I am indebted to Dr. W. Gill Wylie, of New York, who gave me the autopsy material with the following clinical history:

"Mr. Van D—, a large, robust, healthy man, denying syphilis, had not been sick except with chills and fever, until four or five years previous to his death, when he gradually became uncertain in his gait, and began to lose the proper co-ordinative use of the muscles of the legs. Very gradually this condition grew worse, so that he could not stand upright very well and his arms became ataxic. Vision became impaired and he could see with only the lower half of the retina. He did not suffer pain. The bladder, bowels and digestion remained in good order until a short time before death. During the latter stages of the illness, the man had at times *attacks of choking*. He finally became insane, and *died in one of the choking attacks*. In this fatal attack there was apparently a paralysis of the laryngeal muscles."

Microscopical Examination.—The brain and first two segments of the spinal cord were removed at the autopsy. Sections of the cord show the characteristic lesions of tabes in the posterior columns. The sclerosis cannot be traced

* Read at the New York Neurological Society, November 5, 1889.

upward beyond the point where the nuclei of the posterior columns become well developed. The nuclei of spinal accessory and vagus, and the ascending roots of the vagus are normal. The ganglion cells and fine nerve fibres in the motor cortex are normal.

The root fascicles of the vagus and spinal accessory nerves on both sides were removed from the medulla and examined separately. Sections of these fascicles, (stained double with hæmatoxylin and acid fuchsine, and by Weigert's method) show the lesions of a chronic diffuse neuritis, —areas of neuroglial increase with the atrophy of the nerve fibres. There is an increase of nuclei in the sclerotic portions of the fascicles, and a few small round cells lie about the blood-vessels in one or two places in the damaged portions of the fascicle. (Some caution is required in deciding about degenerated areas in the vagus and spinal accessory nerve roots because both of these roots contain bundles of fine medullated fibres destined to pass to the sympathetic system; these fibres have a more abundant connective-tissue matrix than the surrounding fibres, and in oblique sections, stained by Weigert's method, bundles of these fibres look like sclerotic patches). The neuritis does not affect all of the fascicles uniformly, some are but very slightly involved by it, and the roots of the right side are much more extensively involved than those of the left side. The hypoglossal and trigeminal roots are normal except that in the latter a few small round cells accompany some of the coarser endoneural septa.

Laryngeal crises in tabes were first described by Fereol¹ in 1876, and Cherschvesky² in 1881 collected 18 cases and divided them, depending upon the duration of the attack, into mild cases, lasting about 90 seconds; medium cases, lasting from five to ten minutes; and severe cases, lasting several hours. The mechanism of these crises is not uniform in all cases or all stages of the attacks, they occur in two ways. An analysis of the published cases admits of their division into two forms: 1. A spasmodic form. 2. A paretic form.

In the first form, some of the attacks, probably as a rule

the earlier and less severe ones, are of a purely spasmodic character. The larynx is perfectly normal and the crises result from a contraction of the glottis abductors. Attacks of this kind, which may last for months or years are well illustrated by Landgraf's³ case, which was under careful observation several months, and the author using the laryngoscope, was assured of the reflex spasmodic nature of the crises. McKenzie³ and Cherchvesky² each give two cases of crises in which the larynx was normal.

The second form of the laryngeal crises includes the majority of the recorded cases. The crises in these cases are reported as due to a paresis or paralysis of the abductors. This is the so-called posterior paralysis of the Germans—*posticus-lähmung* Gerhardt. (Virchow's Archiv., xxiii., 1863). The writer thinks that in the great majority of these cases of posterior paralysis producing crises, are not due to the exclusive paralysis of the abductors, as is intimated by the term *posticus-lähmung* or posterior paralysis, but are due to a weakening or paresis of *both adductors and abductors*, and that in this paresis of both sets of glottis muscles the *action of the abductors* is impaired more than that of the adductors. How the function of the abductors becomes more impaired than that of their opponents in paresis of both sets of muscles, so that in cases of laryngeal crises, the abductors appear to be exclusively paralyzed, may be better understood by considering the experiments of Semon and Horsley, Hooper and Donaldson.

Semon and Horsley (Brit. Med. Jour., 1886, p. 405) have shown in a large number of experiments on dogs and monkeys (20 experiments stimulating the uncut nerve; 12 experiments stimulating the divided nerve), that electrical stimulation of the recurrent laryngeal nerve quite uniformly caused adduction of the glottis, except when the animals were deeply narcotized. The same result occurred even when the larynx was removed from the animal. These experiments show that although the crico arytenoidei postici are larger than any other of the intrinsic laryngeal muscles, yet under ordinary circumstances, they are not powerful enough to resist the combined forces of their opponents,

the adductors. When animals are deeply narcotized, stimulation of the recurrent produces the opposite effect—abduction of the vocal cords. (Hooper, *N. Y. Med. Jour.*, July 4, 1885; June 6, 1886, Semon and Horsley, *l. c.*); Donaldson, (*Amer. Jour. Med. Sci.*, July, 1886), operating on dogs, found that the abductors responded to a weaker current than the adductors; an initial weak current in the recurrent nerve becoming gradually stronger produced at first abduction, which gradually gave way to adduction of the cords.

A peculiar feature of the posterior crico arytenoidei, discovered by Semon and Horsley, is that these muscles lose their electrical excitability long before any of their antagonists, when the laryngeal muscles are individually stimulated. They become fatigued and die sooner than the other muscles. In other words, a greater degree of destructive metamorphosis is coincident with their activity. A still farther distinction between the abductors and the adductors is the morphological distinction pointed out by Simonowsky, (*Inter. Centralbl. f. Laryngol.*, Vol. ii., p. 346), and Gruetzner (*Breslauer Aertz. Ztschrift.*, 1883, No. 18), that the abductors belong to the red muscles of Ranvier; whereas the adductors, at least the thyreo-arytenoideus, belong to the so-called white muscles. (This histological distinction however is not definitely settled, for the researches of these authors were incomplete when published).

Semon, (*Berlin Klin. Wschrft.*, 1883, No. 46; *Archiv. of Laryngol.*, Vol. ii., p. 197), from a study of laryngeal paralyses of neural origin, made the statement that in cases of injury or organic disease of the centres or trunks of the motor laryngeal nerves, the abductor muscle or muscles succumb first. (Semon's statement is also supported by Rosenbach, *Breslauer Aertz. Ztschrft.*, 1880, Nos. 2, 3; *Berlin Klin. Wschrft.*, 1884, No. 17; *Virch. Arch.*, Bd. 99). The difficult thing to understand about Semon's statement is why the abductors should be thus selected when the same motor nerve and nucleus supplies both adductors and abductors. Why, for instance, should a chronic neuritis of the roots or trunk of the vagus containing the motor fibres

for all of the laryngeal muscles (except the crico-thyroids) cause an earlier or more extensive paralysis in the abductors than in any of the other muscles?

Unless the motor fibres of the abductors are relatively much more numerous than the adductor fibres—and this is not likely to be the case for the abductor group of muscles is more voluminous—there is no reason why a chronic neuritis of the vagus roots should not involve the adductor nerve fibres just as much as the abductor fibres. Probably the truth of the matter is, that with such a lesion, the abductors suffer no greater loss of power than the adductors, but as the direction of the fibres in the abductors—the posterior crico-arytænoidei—seems to put them at a greater mechanical disadvantage in moving the arytænod cartilages than the lateral muscles; a uniform diminution of absolute power in both the adductors and abductors, may produce a far greater impairment of the abductors than of the adductors. Experiments show that the equilibrium of the glottis inclines in favor of the adductors in the normal larynx (except in conditions like ether narcosis), and if the adductors are stronger in the normal larynx under ordinary circumstances, we should expect their strength still more prominently asserted in the disturbed equilibrium of the glottis, when both sets of muscles are paretic, for the subtraction of a given amount of absolute power from each set of muscles would diminish the action of the abductors in a greater ratio than the adductors, which are larger, stronger, and work at the glottis from a better mechanical standpoint.

Thus it may be that the predilection of the abductors to become paretic, as observed by Semon, is only apparent, and, as Gowers (*Dis. Nerv. Syst.*, 1888, p. 692) suggests, it would be better to speak of this condition as loss or impairment of abduction, rather than as paralysis of the abductors or posterior paralysis, in the sense that the abductors are exclusively paralyzed.

Believing that a good many of these so-called posterior paralytic forms—the second form of the crises—are caused by a chronic neuritis of the motor laryngeal fibres in the roots (or trunk) of the vagus, it seems that they occur in

this way. The neuritis causes a (nearly equal) reduction of absolute power in both the constrictors and dilators of the glottis, which renders their equilibrium so unstable that with the slightest reflex or direct motor impulses the adductors, working on a superior mechanical vantage ground, gain the ascendancy, and a crisis results. In other words, the exciting cause of many of the crises of this form, is a reflex or direct motor sent through the recurrent nerve to a paretic, unstable group of antagonistic muscles with the balance of power in favor of the adductors, operates like the stimuli of the recurrent nerve in the experiments of Semon and Horsley. This excites a spasm of the adductors which is intensified or prolonged and induced the more readily perhaps, from the impairment of the function of the abductors being greater than that of the adductors, although both sets of muscles are paretic to a nearly equal extent. When the adductors become fatigued by their tonus, or should a condition like ether narcosis occur from the attack, the crisis gradually ceases.

Such an explanation of this second form of the crises accounts for the sudden way in which they occur as attacks, and is supported by the fact that deadening the reflexes from the fauces pharynx and larynx by cocaine and bromides are of benefit in many cases.

Changes in the nerve roots and in the peripheral nerves are so frequently present in tabes, and central lesions in the medulla are so comparatively infrequent, that probably chronic neuritis of the roots—analogueous to the neuritis of the spinal nerve roots—or trunks of the laryngeal nerves is quite uniformly the lesion producing the laryngeal crises. The neuritis of the roots of the tenth and accessory portion of the eleventh nerves in this case explains very well how the laryngeal crises of both kinds may occur, if some points in the laryngeal innervation are referred to.

The superior laryngeal nerve is the sensory nerve of the larynx, and is composed as a rule entirely of fibres from the accessory portion of the spinal accessory. Vrolik (Kirke's Handbook of Physiology, 1889, p. 614), found in the chimpanzee that the internal division of the spinal accessory

passed directly to the larynx without fusing with the vagus. The supposed motor supply of the superior laryngeal nerve to the crico-arytænoidei postici is denied by Schech (*Lit. of Biol.*, ix., p. 258). Stimulation of the central end of the superior laryngeal produces decided closure of the glottis (Krause, *Virch. Arch.*, Bd. 98). The recurrent laryngeal nerve, supplying all of the muscles except the crico-thyroids, as a rule derives its motor fibres from the vagus; occasionally this nerve varies in its composition, and its motor fibres come partly from the vagus and partly from accessory vagus. The recurrent nerve also contains a few sensory fibres, the centripital path of which is not definitely known.

The statements of Bischoff, Longet, Bernard and Morganti (references given in Donaldson's paper), to the effect that the accessory portion of the spinal accessory is principally a motor nerve are quite untenable at present. If the superior laryngeal was chiefly motor, stimulation of its central end ought not to produce such a pronounced reflex closure of the glottis; this experiment indicates that the superior laryngeal is more a sensory than a motor nerve. A study of the central termination of the accessory portion of the spinal accessory seems to show that it is mainly a sensory nerve. Recurrent motor fibres like those of the vagus passing to the nucleus ambiguus may be seen in the adult medulla, passing in from the very uppermost roots of the accessory portion; but as the sections pass down to the middle and lower levels of the roots of the accessory portion, these recurrent fibres become fewer and fewer and disappear, so that it is doubtful if recurrent motor fibres like those of the vagus, exist in the middle and lower roots of the accessory portion. I think from this that all of the root strands of the accessory portion are sensory fibres from the larynx and pharynx, except the uppermost strands nearest the vagus, which contain motor fibres for the crico-thyroids and inferior constrictor, and a vicarious motor supply for the pharyngeal and recurrent branches of the vagus, which nerves seem to be subject to occasional variation in their composition, as they sometimes have a portion of their

usually almost exclusively vagal supply of motor filaments replaced by motor fibres from the accessory portion.⁴

A chronic neuritis of the roots (or trunk) of the accessory portion of the spinal accessory, irritating the laryngeal sensory fibres, would be responded to by motor impulses passing down the vagus which would cause, as in electric stimulation of the motor laryngeal fibres, adduction of the cords. This lesion would account for the first form of the crises. If this neuritis should be sufficiently destructive anæsthesia of the larynx would result. Krause and Fränkel each report a case of anæsthesia of the larynx in conjunction with tabetic laryngeal crises.

An early irritative stage of the chronic neuritis in the vagus roots (or trunk) might also cause crises of the first form, by stimulating the motor laryngeal fibres and producing direct spasms of the adductors. A later destructive of this neuritis might induce paresis or paralysis in both the abductors and adductors. But with this condition abductorial impairment is greater than adductorial, and this coupled with the experimental facts that the abductors are unable to resist the forces of their antagonists in the normal larynx and become fatigued sooner, suggests as an explanation of the second form of crisis, that at certain times with reflex or direct motor stimuli, (which might not be of sufficient intensity to provoke a spasm in the normal larynx), the adductors get the balance of power and their tonus may be enhanced by the greater degree of metabolism in the abductors. When both the vagus and accessory vagus are diseased together, the conditions are favorable for the production of violent crises of the second form.

Review of Some of the reported Cases of Laryngeal Crises in Tabes.—Krause⁵ collects eight cases which were examined laryngoscopically. The vocal cords were quite uniformly in the median or adduction position. Three of Chervinsky's cases had paralysis of the left cord, and in one case there was paralysis of the abductors on both sides.

⁴ References to other less definitely settled points in laryngeal innervation are given by Lennox Browne, *The Throat and its Diseases*, 1887, p. 477.

Wegener's two cases had paralysis of the left abductors. Fränkel's⁶ case had paralysis of one cord and paresis of the other. Fournier (*Leçons sur le période prae ataxique de tabes d'origine syphilitique*, Paris, 1885), reports five cases of one-sided laryngeal paralysis in tabetics who had no crises. Kahler's⁷ case had paralysis of the left cord, and crico-arytænoideus posticus and dysphagia. Crises in this case could be elicited at times by causing the patient to swallow a glass of water.

Krause⁸ reports two cases, in one of which anæsthesia of the larynx was present, but there was good laryngeal reflex irritability so that every touch of the larynx with the sound elicited a crisis. In the second case laryngeal irritability was absent; the larynx could be sounded without awakening crises. In both cases crises could be produced by irritating the nasal fossæ. Oppenheim¹⁰ presented two cases having gastric crises. In one of the cases, the crises was of a purely spasmodic character for several years, but subsequently the crises assumed the features of the second form associated with loss or impairment of abduction. The voice became rough and the crico-arytænoidei postici and the thyro-arytænoideus became paretic. Pulse anomalies were also present in this case.

In the second case, the crises were very violent and one of them proved fatal. (Microscopical examination in this case showed neuritis of the vagus and accessory vagus roots) Besides the fatal crisis recorded in this paper, a third fatal crisis is instanced by Keller, (quoted by Cherchvesky) Krishaber¹⁵ had to do tracheotomy in an urgent case of tabetic laryngeal crises.

In a third case of Oppenheim's¹¹ there were spasmodic pharyngeal attacks followed by movements of swallowing. A fourth case, by the same writer, had paralysis of the right cord which remained immobile when the right recurrent nerve was stimulated electrically, while the left cord responded to stimulation of the left recurrent.

Landgraf's³ case of purely spasmodic crises of the first form is very interesting in connection with the fact, that two years afterward the patient developed paralysis of the

sterno-cleido-mastoid and trapezius on one side. (Reported by Martius, Berlin Klin. Wochenschrift, No. 8, 1887). This would indicate that the spasmodic reflex crises were caused by a neuritis of the accessory portion of the eleventh nerve, which subsequently extended to the spinal portion of the nerve. McBride's¹³ case of tabetic crises also had paresis of the sterno-cleido-mastoid and dysphagia, frequent heart action, and finally Cheyne-Stokes respiration, indicating neuritis of both the vagus and spinal portion of the spinal accessory.

In Weil's¹⁴ case, the crises, which as a rule do not appear until the tabetic symptoms are well established, were the initial symptoms of the disease, and enhances the suggestion of Buzzard and Semon, of testing the knee-jerk in all cases of laryngeal paralyses. Weil regarded the case as one of posterior paralysis. (Jeleneffy, Berlin Klin. Wochenschrift, 1888, p. 728, reviewing Weil's case, and finding that the crises were often originated by peripheral stimuli, questions whether there really was posterior paralysis). Weil instances Krishaber's,¹⁵ Lhoste's¹⁶ and Morgan's¹⁷ cases as of posterior paralysis similar to his own.

Some writers have reported cases of laryngeal cases due to ataxia of the laryngeal muscles. The consideration of ataxia of the glottis muscles is exceedingly complicated. A central lesion would be indicated in laryngeal ataxia rather than lesions in the roots or trunks of the laryngeal nerves, which are believed by the writer to be the cause of the crisis in the majority of the cases. If such cases of laryngeal ataxia-producing crises exist, it must be very difficult to distinguish them from the other two forms alluded to in this paper.

Summary of the Lesions in the Reported Cases.—Jean¹ found atrophy of the left vagus, accessory, and recurrent laryngeal nerves and left thyro-arytænoideus. Kahler thickening of the ependyma involving the right vagus nucleus, and principally the nucleus of the right accessory vagus nerve. (The paralysis in this case could be explained by the damage done to the recurrent or motor fibres, passing to the nucleus ambiguus, by the lesion in the sensory

vagus nucleus.) Demange¹⁹: lesions similar to those in Kahler's case. Landouzy and Dejerine: atrophy of the vagus roots and nucleus of the vagus accessory nerve. Oppenheim: neuritis of the vagus and accessory vagus roots and degeneration of the recurrent laryngeal nerve.

The *pharyngeal crises*—dysphagia, with or without spasmodic deglutitory movements, which occasionally are in company with the laryngeal crises—may be accounted for by a chronic neuritis of the roots (or trunks) of the ninth, tenth, or accessory portion of the eleventh nerves; but it is difficult to explain these crises in detail, owing to the complicated and apparently variable distribution of these nerves to the pharyngeal plexus. The simplest account of the pharyngeal plexus is given by Schwalbe ("Lehrbuch," 1878, p. 874). All three of the nerves of the eighth pair send both sensory and motor branches to the pharynx. The principal sensory conductor is the glosso-pharyngeal, which also sends motor fibres to the stylo-pharyngeus, and middle constrictor. The principal motor nerve is the vagus. The accessory fibres to the pharynx are sensory, except those to the inferior constrictor, and except when accessory fibres replace the motor vagus fibres in the pharyngeal branch of the vagus. A chronic neuritis in the roots or trunks of one or more of these nerves might produce reflex or direct pharyngeal movements, or a degree of paresis of some of the muscles, giving rise to dysphagia.

The *gastric crises* might occur in two ways: Reflexly—for instance, by a neuritis involving the sensory pharyngeal filaments; directly, by a neuritis of the upper three cervical nerve roots, or of the vagus accessory roots or of the trunk of the vagus, which is the path of the visceromotor fibres upon which depend peristaltic contraction of the thoracic oesophagus, stomach, and small intestines (Gaskell, "Jour. of Phys.," vol. vii.).

The *bronchial crises*—spasms of the larger bronchi, with attacks of rough coughing—might be caused by a neuritis of the vagus which carries bronchial sensory fibres, and the visceromotor fibres supplying the smooth muscles of the bronchi.

The *cardiac crises*—anginoid attacks, and the pulse anomalies occurring in tabes—might be caused by a vagus neuritis interfering with the cardio-inhibitory fibres. Cardiac disturbances might also occur from the cord lesion in the upper dorsal region, whence, according to Gaskell, the cardio-augmentor fibres issue in the anterior roots.

The reason of the other tabetic visceral symptoms may be made clearer by referring to the researches of Gaskell (*loc. cit.*) on the distribution and origin of the sympathetic fibres. The fibres of the sympathetic arise in the spinal cord and lower medulla, and issue thence, as finely medullated fibres in the anterior and posterior roots, in three great channels, viz.: The cranio-cervical outflow, in the vagus, in the spinal accessory passing to the vagus through the ganglion trunci, and in the upper three cervical nerve roots; the thoracic outflow, situated between and including the second dorsal and second lumbar nerve roots; the sacral outflow, from the second and third sacral nerves. The cranio-cervical outflow contains the cardio-inhibitory fibres and the visceromotor fibres for the stomach and circular fibres of the small intestine. The thoracic outflow passes upward to the cervical sympathetic trunk and ganglia, medially to the prævertebral ganglia (semilunar and mesenteric) and splanchnics, and downward to the hypogastric plexus. The thoracic outflow contains the visceromotor nerves for all parts of the body, glandular nerves, visceroinhibitory nerves, cardio-augmentor nerves, and visceromotor nerves for the circular fibres of the hind-gut, and some of the vaso-dilator fibres. The sacral outflow, composing the nervi-erigentes, consists of fibres passing directly to the hypogastric plexus, whence they pass upward to the inferior mesenteric ganglion and downward to the bladder, rectum, and generative organs. The sacral outflow contains vaso-dilator fibres for the penis (stimulation of the anterior second and third sacral roots produces erections in rabbits: Gaskell, "Jour. of Phys.," vol. viii.) and vaso-dilator fibres for the lower extremities. This outflow also contains the visceromotor fibres for the bladder, uterus, and longitudinal muscles of the rectum.

These three regions of the cord, whence issue the finely medullated fibres to the sympathetic system, correspond so closely with the situation of the cervical nucleus, the columns of Clark, and the sacral nucleus, that Gaskell thinks that the central origin of the sympathetic is partly in these nuclei. As the central tabetic lesions, or neuritis of the spinal nerve roots, may involve these portions of the cord, we may try to explain how some of the other visceral and trophic symptoms in tabes arise.

The tabetic lesion in the second and third sacral segments, interfering with the vaso-dilator fibres of the sexual organs, would account for some of the sexual disturbances and the *clitoris crises*. The paroxysms of rectal pain and tenesmus—*rectal crises*—if not caused by the lower dorsal portion of the cord lesion, involving the visceromotor nerves of the circular rectal muscles, may be caused by the sacral portion of the cord lesion interfering with the motor nerves of the longitudinal muscle layer of the rectum. The *vesical disturbances and crises*, the *urethral crises*, and the weakness of the rectal and vesical sphincters may be caused by the lesion in the second and third sacral segments involving the visceromotor fibres of these organs.

As the vaso-dilator and vaso-motor fibres to the lower extremities pass out of the second and third anterior sacral roots and lower portion of the dorsal region respectively, lesions in these portions of the cord may account for the trophic disturbances in the lower limbs, viz.: Brittleness of the bones, atrophy of their heads, etc., arthropathies of the knee-joint, changes in the tarsal bones and joints, flattening the foot (tabetic foot of Charcot and Féré), temporary œdema without renal lesions, local sweating, perforating ulcer of the foot, unprovoked ulceration of the toes, bad growth of the nails, etc.

Intestinal troubles are manifested by constipation, spasmodic attacks, and peculiar diarrhœal attacks, which Pierret supposed to be of vaso-motor origin. If not due to a vague neuritis, the intestinal symptoms may be caused by the lesion in the dorsal cord. The constipation may occur from a destruction of some of the visceromotor fibres to the gut.

The dorsal region of the cord probably contains the vasodilators of the guts.

The lesion producing the *nephritic crises* is difficult to locate. The path of the visceromotor fibres to the ureter is not known. According to Bradford ("Journal of Phys.," vol. ix., p. 358) the renal vaso-motor fibres issue from the cord in the ninth, tenth, and eleventh anterior dorsal roots. The lesion in this portion of the cord might interfere with the renal circulation.

LITERATURE OF CASES OF LARYNGEAL CRISES IN TABES.

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¹⁹ Demange. — Chute spontanée des dents et crises laryngées et gastriques chez les ataxiques. Revue de Méd., 1882, No. 3.

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²² Rummo. — Sur un cas non commun de tabes vulvaire primitif. L'Union Médicale, 1884, No. 81.

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Periscope.

By LOUISE FISKE-BRYSON, M.D., AND A. FREEMAN, M.D.

MELANCHOLIA, FROM THE PHYSIOLOGICAL AND EVOLUTIONARY POINT OF VIEW.

The "Journal of Mental Science," January, 1890, contains George M. Robertson's paper upon this interesting subject. Probably no form of insanity includes so many varieties, or is characterized by such peculiar and even contradictory symptoms as melancholia. The excitement may, at times, equal that of acute mania, or the passivity may be that of absolute inertia. What explanation is there of these diverse symptoms in melancholia? In answering this question, the fact must be borne in mind that the investigation concerns the functions of an organ which has become diseased—the brain—the function in this case being the production of depressed or painful emotions. To understand aright this abnormal function, a careful study of depressed or painful emotion in a healthy brain is the first step. Health (Hamilton) has been defined as "that condition of structure and function . . . which we find to be commonest," and disease as "any departure from the normal (or healthy) standard of structure or function." Health and disease are relative terms, without a sharp dividing line. Most of the symptoms of melancholia can be traced to some normal function. Darwin has shown that many of the expressions of emotion in man are the modifications of similar expressions in the lower animals. If reversion to older and lower types takes place, the expression of emotion in diseased states may acquire a closer resemblance to the same emotion in the lower animals. A light appears to be thrown on some of the symptoms of melancholia by this assumption.

Depressed emotion in health comprises at least three important elements: a characteristic feeling of depression or pain, the train of ideas associated with this feeling, and the special relationship to different bodily organs, particularly the muscular system. Depressions may range from sensations of slight uneasiness or unhappiness to those of fright and the most abject terror. In depressed emotion, there is a sudden interruption to the normal flow of ideas,

and thoughts are stimulated which are all of a gloomy or disagreeable nature. Owing to this fact the judgment is impaired. If the sudden interruption to the flow of ideas be great, temporary confusion may result. The most wonderful and characteristic accompaniments of the emotions, however, are the changes in the bodily organs, including the muscular system. These organic changes are not merely the expression or effect of the emotion. They are its "material cause and support." Experimentally, this receives confirmation from observations in hypnotism. Organic changes in depressed or painful emotion extend over a wide range and include voluntary muscles—thus tone being increased or diminished—and involuntary muscles, like those of the heart, intestines, etc., together with vasomotor, respiratory and secretory disturbances.

It is possible to be greatly depressed, to find no delight in man, or woman either, to think nothing worth while, and to desire annihilation without being insane. The Rubicon of insanity is, however, crossed when once the judgment—that is, the ideational element of the emotion—becomes much impaired. The symptoms then differ in marked degree from the normal. Thus, a man suffering from deep sorrow cannot carry on a conversation, cannot think, and holds biased opinions. The condition may be only temporary. When carried over into the domain of disease it is called passing melancholia. If ideation becomes more impaired, he passes from the holding false opinions to the state of entertaining delusions; and if these refer to phenomena outside himself, the condition is one of delusional melancholia. If, added to the impaired judgment, the want of pleasure in organic and other sensations and the feeling of bodily ill-being become more marked, and if the delusions are concerning the patient's bodily health, the condition is that of hypochondriacal melancholia. When some one delusion concerning bodily health, usually an abdominal organ, becomes a fixed idea and is regarded with profound sorrow as the cause of all depression and woe, the variety of insanity it represents has been called visceral melancholia. If, instead, the wish to be dead and away, with occasional thoughts of suicide, there is a constant desire on the part of the patient to kill himself—whether founded on a delusion or in order to escape present intense misery—the variety is termed suicidal melancholia. Here, then, are five clinical types of depression in abnormal function of the brain. In none of these five types has any new symptom been suddenly sprung upon us; all the symptoms are seen to be developments of phenomena which exist in health, from which their origin is obvious.

Active melancholia, of which the chief symptoms are motor excitement, resistance, violence and confusion, bears a close relation to normal terror. The frightened child gives a loud, high-pitched scream, and the breathing becomes agitated. The eyebrows are drawn up, the eyes stare and protrude, the mouth is open, the facial muscles are tensely contracted, and even the hair may stand on end. General muscular tension is very great and the child may run away. If caught by the object of its terror, it fights, struggles and screams with extraordinary violence till exhausted. The disturbance to the train of ideas is very great, and mental confusion and want of judgment prevail. Similar conditions prevail in timid lower animals, as birds and rabbits. From the motor point of view, melancholia with stupor has been defined as "more or less complete loss of reaction to stimuli." (Savage). There is a *vasucio* expression, the patient standing or sitting for hours in the same position, taking no notice when spoken to, and without interest or desire of any kind. The hands and feet are cold. This corresponds with that state in health when persons say they are paralyzed with fright, and are powerless to move, even for self-preservation. It is commonly seen in lower animals, as where a rabbit lies on its side and allows a weazel to approach it. The so-called charm exercised by snakes over birds is believed also (Faytele) to be an example of it. Intense fear usually exists, but not always. Dr. Livingstone felt no pain or fear, but was paralyzed when in the clutches of a lion.

The chief varieties of melancholia—passive, active, and with stupor—though dissimilar, but show their relationship by a tendency into one another. Having studied them physiologically, their consideration from the evolutionary point of view is next in order, to explain their primary origin.

The finer and more complex emotions are developed from the coarser and more simple. Anxiety is only fright spread out thin (Hughlings-Jackson). Fear or fright in unevolved man or lower animals is the origin of depressed emotion. The struggles and combats of mankind's remote ancestors in the distant long agoes have given the prostration, pallor, perspiration, trembling, or complete relaxation of the muscles that to-day mark normal or abnormal depression. In those emotions produced by anticipations of future misery, there is increased tone and excitement; in those which dwell upon the past, there is relaxation. By action and resistance we may surmount danger; hence, increased tone. When the time for action is passed, or

action is useless, there is relaxation, despair. The tension of the muscles in active melancholia takes the form of resistance, and usually combined with it is the allied mental condition of obstinacy. The patients seem to resist on principle anything and everything that is done for them. Their perpetual meaningless resistance can only be explained on the struggling animal theory.

Violence in melancholia may be perpetual or sudden; and the tendency of melancholiacs to strike when thwarted closely resembles the actions of a struggling animal. A desire to kill is not surprising, if evolved from a condition in which one animal is doing its utmost to kill another. Restlessness, aimless wandering, or a desire to run away, are expressions similar to those of wild animals in menageries, who walk up and down their cages. Then there is the same mournful standing in one place for a long time, after the fashion of caged creatures.

Darwin states that when suffering severe pain, groaning brings relief, by letting off energy; and after the habit is formed, one groans reflexly, by association of painful states, in mental depression. Sufferers from melancholia often render day and night hideous by their shouts and cries, which are probably derived from the scream of terror, the frightened call for help, that is common to man and lower animals. The staring eyes also resemble those of a frightened animal, which opens the eyes widely, that there may be no obstruction to vision, in order to discover the enemy or means of escape. The melancholiac is also selfish, like an animal struggling for existence, who thus is looking out for number one in a pre-eminent degree. Destructiveness, cruelty, indecency, untruth, cannot be explained satisfactorily on the ground of mere depression. But regarded from the standpoint of the evolution of emotion in an animal struggling for its life, bent on doing injury, and exercising no self-control, these things are better understood. The coldness of the extremities and feebleness of circulation is analogous to the muscular and cardiac exhaustion which succeeds a mortal combat. The drooping of the eye-lids, the general languor and paralysis of the sphincters, is due to the same collapse. Refusal to take food is common to birds and animals in captivity, and is probably owing to depressed emotion. Bristling of the hair, which may occur in a small percentage of cases, and a downy appearance of the face, owing to the fine hairs being erect, are manifestations analogous to the erection of the hair or feathers of a frightened animal, in order that it may appear larger and more dangerous to its assailant. Fear increases the amount and watery constituents of urine.

Diabetes insipidus existed in one melancholiac under observation. In terror there is also an increased secretion of sweat. This has not been observed in melancholia. Catalepsy, comparatively rare in melancholia, the author thinks is derived physiologically from the "fixation" of surprise. For example: when eating, and in the act of raising a spoon to the mouth, some occurrence may cause surprise, and the individual will hold the spoon in a fixed position for a considerable time near the lips. In this fixation, Robertson thinks, we have the physiological elements of catalepsy. In the lower animals this tendency has been highly developed by natural selection, having proved useful under certain circumstances, and is the real explanation of the so-called simulation of death by insects and animals.

Darwin investigated this question of shamming death among insects, and after many observations found that in no one instance was the attitude similar to that of a dead insect of the same species, and in many cases it was very unlike. He came to the conclusion that the position was due to the paralyzing effects of excessive fear. There is probably no conscious simulation of death; the condition may be due to an exaggeration of the fixation in surprised fright. It occurs in nearly all classes of the animal kingdom (Romanes), probably best seen in mice. That this condition is a cataleptic stupor produced by fright is believed by several eminent comparative psychologists (Romanes, Preyer, Duncan, Couch). There is also a connection between this condition in the lower animals and catalepsy in melancholia, in a number of recorded cases of sudden catalepsy produced by fright, may be regarded as bridging over the borderland of health and disease.

Nightmare is also probably analogous to fright, rigidity, and catalepsy.

NEURASTHENIA OR ANÆMIA.

In *La Province Médicale*, Feb. 22, 1890, L. Bouveret states, careful discrimination is sometimes necessary to avoid mistaking one of these conditions for the other, for many physicians are in the habit of calling anæmic all patients who complain of nervous phenomena not attributable to organ nerve-centre abnormality. A large number of neurasthenic men, as well as women, are not in the slightest degree anæmic, the blood in color and composition being perfectly normal. Intense anæmia undermines nerve function; and the functional nerve troubles thus brought about bear a certain resemblance to nerve exhaustion.

Thus it is difficult to make a positively correct diagnosis when the patient is both anæmic and neurasthenic. In such a case there is manifest a marked disparity between the degree of anæmia and the intensity of nervous exhaustion. In cerebral neurasthenia, any intellectual effort produces shortly a sensation of painful exhaustion; yet the patient may endure without too great strain considerable physical fatigue, such as that produced by a long walk. In anæmia, on the contrary, mental and physical fatigue are equally exhausting. The so-called gouty or rheumatic neurasthenics who suffer from wandering pains in the extremities, head, and trunk, these pains being greatly influenced by atmospheric changes and just before a storm, are really neurasthenics pure and simple, as their earlier history and that of their family will abundantly prove. Chronic alcoholism bears some analogy to certain forms of neurasthenia. The insomnia, bad dreams, impairment of the mental faculties, numbness and tingling in the limbs, diminution of muscular force, are phenomena sometimes present in both cases. But chronic alcoholism has facial expression that is animated and gay when contrasted with the depressed and languid countenance of neurasthenia. The digestive trouble is still more distinct, for the gastric catarrh of the drunkard is easily distinguished from the gastro-intestinal atony of the nervously exhausted. When the two depraved conditions—chronic alcoholism and neurasthenia—are combined, the diagnostic situation is complex. But neurasthenic stigmata are not hard to find when the phenomena have a neurotic basis. Neurasthenia and hysteria may be developed by the syphilitic poison. Fournier has described a multitude of nervous troubles, especially among women, that may justly be ascribed to syphilis, especially in its secondary stage. With men the nervous manifestations more often take on a hypochondriacal form. Physical agents—mechanical medicine—are those that cure neurasthenia. Drugs add to the original disease a state of chronic poisoning more difficult to combat than neurasthenia itself.

MULTIPLE NEURITIS FOLLOWING TYPHOID FEVER.

In the Omaha Clinic, January, 1890, Dr. W. O. Bridges reports such a case, the subject of a clinical lecture at St. Joseph's Hospital. The patient had been free from rise in temperature for several days, was up and about the room, when a slight fever returned; and within three or four days he began to experience a weakness of the legs, accompanied by shooting-pains through the muscles below the knees.

This was followed by numbness, tingling, and loss of power. The same symptoms without pain soon developed in the forearms and hands, and within a week the patient lost all power of the extremities below the knee and elbow. At times there was swelling of the hands and feet, with areas of perspiration. The parts affected had been particularly sensitive during his earliest illness. The lecturer gave acute arsenical poisoning, lead, alcohol, acute infectious diseases—as diphtheria, variola, typhoid and typhus fevers—and the action of cold as causes of multiple neuritis, of which diphtheritic and lead paralysis are varieties. The diagnosis he thought sometimes difficult, the disease often bearing marked resemblance to locomotor ataxia, anterior polio-myelitis and diffuse myelitis. The symptoms collectively and the history of the case would clear up any doubt. Iron with *nux vomica* and iodide of potassium were recommended as remedies. Treatment of the muscles would consist of the galvanic current and friction of the surface with passive motion of the joints on alternate days.

SOURCE AND NATURE OF LEAD POISONING.

The "Medical News," March 1, 1890, states that Mr. Herbert Whitby, of England, thinks that a possible source of plumbism in that country exists in the "tons upon tons of lead scattered over the land by sportsmen. In some places on the moors the ground is literally paved with lead, and the effects are terrible to contemplate when it is considered that water from the moors forms part of the water supply of many towns." America is in no immediate danger from a similar cause. The "Clinical and Chemical Observations on Plumbism" by Dr. John Brown, of London, refers to serious outbreaks of lead poisoning during the past few years in the north of England, especially in Lancashire and Yorkshire, where but few towns have escaped when the water supply has been largely dependent upon the rainfall. The author affirms that there is no disease more insidious, ubiquitous, and manifold in its manifestations, and which so closely simulates other diseases as lead. Many cases of so-called meningitis, encephalitis, cerebro spinal meningitis, and epilepsy, if traced to their true cause, would be found really due to lead-polluted water. The clinical features prove conclusively that lead is a nerve poison. The symptoms observed favor the view that it has an elective action on the nerve centres, particularly the multipolar cells—situated in the anterior cornua of the upper

portion of the spinal cord—which control the action of the extensors of the hand. The anæmia, neuralgia, cachexia, paralysis, and peripheral neuritis, are due to defective metabolism which is secondary to the direct action of lead upon the nerve centres.

Dr. Brown suggests the use of substitutes for lead pipes: glass-lined or cast-iron pipes, glazed earthenware, or solid block tin pipes. Tinned or tin-washed lead pipes are *more dangerous* than the ordinary lead pipes. Lead pipe from the virgin ore is safer than tinned lead pipes, or pipes made from mixed lead, which contains zinc, tin, and other metals.

IDEAS OF PERSECUTION IN EXOPHTHALMIC GOITRE.

"L'Union Médical," March 6, 1890, gives an abstract of Ballet's observations upon this subject. Mental disorders, such as maniacal or depressive states have for many years been recognized as accompaniments of exophthalmic goitre. The speaker himself had pointed out at different times various nervous phenomena that may accompany this disease, such as external ophthalmoplegia, simple polyuria, or glycosuria, epileptiform attacks, together with paralysis of the limbs, true epileptic seizures and other disorders of sensibility and motion dependent on hysteria, chorea, du mal comitial, neuroses associated with Basedow's disease. Locomotor ataxia is sometimes added to this list.

A patient presented by Ballet gave evidence of exophthalmic goitre, multiple paralyses of motor bulbar nerves, and marked hysteria. And besides all this, the poor wretch suffered from a peculiar form of delirium of persecution. Suspicious to a degree, believing every one had designs against him, the patient wandered about incessantly. His father, brother, and Ballet, his physician, were particular objects of distrust. Once he had tried to kill his father, and had made an attempt at suicide. Hallucinations of sight, hearing and smell had given rise to these ideas of persecution. Dreams favored their development, as in the case of the insane. There was no evidence of alcoholic or other toxic delirium. The question naturally arose as to whether the disorders of intellect in this case could be classed as evidences of true delirium (systematic chronic progressive psychosis); and answered negatively, on the ground that in true delirium, auditory hallucinations are the rule. Here visual hallucinations predominated. They presented hysterical characteristics, such as multiplicity, mobility, and zoopsia—the patient at times seeing fierce animals of different colors. Hysteria could not be considered the only

cause of his mental condition; for while hysteria may give rise to terrifying hallucinations of sight, it rarely produces ideas of persecution. Something more is needed than hysteria to transform an hallucination into an idea of persecution; and this something is probably the fantastic, bizarre, unaccommodating frame of mind common to nearly all sufferers from exophthalmic goitre. It will be perceived that this disease alone is insufficient for the development of ideas of persecution, with their possible consequences (homicide, suicide). The addition of hysteria and its hallucinations constitute the material out of which ideas of persecution are created in exophthalmic goitre.

HYDROPHOBIA REPORTED AS SUCCESSFULLY TREATED.

In the "Lancet" of March 1, 1890, Dr. F. Lucas Benham gives a *résumé* of certain cases of hydrophobia that have been reported cured and an account of the treatment employed. Bleeding is the first remedy, strongly insisted upon by Rush. Then comes mercury internally and by inunction. Cinnabar, musk, opium, liquor plumbi, arseniate of soda, are other agents mentioned. That these or any measures may be of service, the disease must be recognized early and combated with energy and decision. As to infrequency of the disease, many practitioners may be as unfamiliar with it as Herberden, who lived ninety years without seeing a single case. On account of almost uniform fatality of the complaint, it has been urged that instances of alleged recovery are not genuine. This may be true of some, but not all. Before deciding positively on some of the doubtful cases, it is necessary to find out what are the variations from the typical forms of the disease, and the limits to the period of incubation. The incubation period has been known to vary greatly in acknowledged fatal cases—from eight days or less to five, seven, and ten years. It is sometimes said that these alleged cases are really hysterical simulation. But it is just as probable that the so-called spurious cases that soon get well are really genuine, especially if it can be shown that many genuine cases are cured. Hysterical symptoms and queer mental derangement commonly form part of the manifestations of hydrophobia (hence the old name "canine madness"). While fear and anxiety may lead to insanity, it is unlikely that such peculiar symptoms as those that prevail in hydrophobia, a disease so rapidly fatal, can really be produced by them. Fear and anxiety are symptoms, not causes, of hydrophobia. They did not exist prior to the onset of the

malady. It is well known that the virus of rabies may produce abortive attacks of hydrophobia or other nervous manifestations. Depression of spirits, local pain, and constriction of the throat followed Dr. Adams' inoculation with the virus of a rabid deer in the Pasteur laboratory. Whether there is an idiopathic form of the disease—the so-called “spontaneous hydrophobia”—as fatal as rabies and closely resembling it, remains a question difficult to decide. L.F.B.

THE PATH OF MOTOR AND SENSORY REINFORCEMENTS.

Dr. S. Weir Mitchell said that since the discovery of sensory and motor reinforcements he had been constantly on the outlook for chances to study the tracks followed by the impulses aroused by these agencies. Such opportunities he considered rare, and perhaps neglected, for neurologists hardly realize this important means of examining muscle reflexes. The histories of two cases were detailed. The first was that of a female, aged 37, who exhibited a faint bluish appearance on the gums, but there were no positive evidences of lead poisoning. She had suffered from difficulty in walking, numbness of the legs and pains in the back and thighs, with some hyperæsthesia of the skin. When first seen her condition was somewhat as follows: legs slightly œdematous, inability to completely control bladder or rectum or to stand alone. Has occasional tremor of the legs. Epigastric reflex present—sole — Patellar reflex increased. Bending great toes causes contraction of quadriceps femoris, and at the ankle there is clonus on slight flexion and also at knees by pulling patellar. The leg fails to drop when supported at the knee, and when bent exhibits the “lead pipe condition.” With knee bent a slight blow on the patellar may give no response, but on clutching the hands a marked one is obtained. Both hands give this reinforcement and also contraction of facial muscles. There is no reinforcement by pain, cold or heat applied to the legs or arms. The history, he said, was that of a spinal sclerosis involving the direct anterior columns. By exciting the knee-jerk feebly voluntary acts of the hand or face increase it; hence the channels by which reinforcements attain to the ganglia involved were open. The volitional force could not reach these centres but the reinforcements can.

The second case was that of a girl aged seven, who after a fall, had paralysis of the legs, bladder and rectum. There was slight knee-jerk in the legs, reinforced by a grimace, clenching the hands or a pin prick. No ankle clonus. Later on knee-jerk was obtained in the right leg,

reinforced by sensory and voluntary motion. This patient was treated with carbonate of ammonium and digitalis, followed by cod liver oil and syrup of the iodide of iron. Extension in the bed was also employed, and under this treatment sensation was greatly improved. Dr. Mitchell remarked that in these cases the chief differences were that in the first motor reinforcement is possible, while sensation failed, and in the second the way for sensory and motor reinforcements is open. Though the will channel be shut, a reinforcement channel is open, which he considered offers some hope for ultimate cure. ("Medical News," March 22, 1890).

THE INSANE IN MASSACHUSETTS.

In a paper bearing this subject, Dr. A. R. Moulton endeavors to show on the one hand that the insane in the hospitals and asylums of Massachusetts, numbering about 5,000, are to a large degree wards of the State, and receive humane treatment; while on the other hand, the hundreds in almshouses and substitutes are defenceless, with no one to speak for them, and whose unfortunate condition is due to a bad system. As a side issue, he says that a small number of unqualified people are trying to maintain themselves by the care of the mentally sick. He asks for suggestions as to how best to meet these conditions. ("Boston Med. & Surg. Journal," March 20, 1890).

REFLEX NEUROSES.

M. A. Starr, M. D., Ph. D., in writing on the above topic, says that the grey matter of the nervous system is collected into three masses: the spinal and medullary centres, the basal ganglia, the cortex. The function of the first was reflex action; of the second, automatic action; and of the third, conscious and voluntary action. Anything increasing the normal inhibitory action of the higher centres or removing it might bring about disturbances of reflex action. The reflex power may be interfered with in several ways: thus, strychnine exaggerates it; it may be suspended by sudden violent irritations, as occurs in severe injuries of the abdomen or head; moderate simultaneous irritations may impair it. The arrest of reflex activity might be voluntarily produced by intention, or involuntarily by cerebral hæmorrhage or fright. The act of control should be constantly exerted to preserve the balance of power and proper harmony of

action in the nervous system. A proper harmony and succession of reflex acts with some definite order is necessary, that the nervous system may fulfill its duty. In the reflex neuroses where there is no defect in reflex action, but only inharmonious reflex activity, the origin of the disease is either in an excess of activity in the lower centres, or a defective control in the higher ones. In the reflex neuroses from supposed peripheral irritation, it is Dr. Starr's opinion that when this is the actual cause, nature attracts attention to the seat of irritation by pain or discomfort. And when the source of irritation is not evident, the probability is that the condition is a manifestation of a defect of control by higher centres, due to impairment of their nutrition and activity. The symptoms in spinal concussion appear to be caused by mental shock, for in railway accidents the passengers who are asleep seldom suffer from after effects. Hypnotic suggestion proves that many so-called spinal neuroses are due to defective cerebral control. In so-called uterine reflex neuroses, there is a general nervous exhaustion due to peripheral pain. The special reflex centres have no part, or very little, in the complex of symptoms. The same, he said, was true of eye-strain. The so-called reflex convulsions from genital irritation in boys, only occur in neurotic children and are preceded by mental irritability. Evidence of spinal irritation in such cases is rare. The same applies to genital irritation in adults. The trouble is *not* spinal or reflex, but cortical. In all these cases, instead of removing the supposed irritation, try to increase the the nutrition of the system to enable it to exert its powers of control and the peripheral effects will disappear. In neurasthenia with peripheral symptoms, in chorea, epilepsy, or hysteria with peripheral irritation, go back of the apparently important symptoms to those of real causal moment, and the therapeutic results will be more satisfactory. ("Medical News," March 22, 1890).

THE INSANITY OF DOUBT.

According to P. C. Knapp, A. M., M. D., this form of insanity may be defined as a mental disturbance, brought on by certain disturbances of the psychical processes, which have been termed insistent or fixed ideas, or imperative conceptions. In the first stage of the disease the patient is susceptible, exacting and timorous, yet has full possession of his reasoning powers. The thoughts become morbid, with a tendency to inquire into the reason for every trifle. In the second stage, he begins to reveal his distress to

friends and requires constant assurance. Finally, he loses confidence in his assurers and the doubts have a still greater dominion over him, although he realizes their unreasonableness. His paper contains some interesting histories of cases of this form of insanity. The disease may begin at any age, and the writer places among the predisposing causes acute diseases or any condition that may weaken the nerve-centres. He says the prognosis is usually considered bad, although numerous observers regard the trouble as a psychical degeneration. Many mild cases recover in three months (Spitzka). The important factor here, according to Dr. Knapp, is hereditary taint, and where this exists the outlook is unfavorable. Treatment should include beside tonics, rest, forced feeding, and especially mental and physical gymnastics. (Reprint from "American Journal of Psychology," 1890.

PROGRESSIVE FACIAL HEMIATROPHY, WITH SOME UNUSUAL SYMPTOMS.

The following interesting case is recorded by B. Sachs, M.D.: E. K., female, nineteen, single; was well until one year ago, when she noticed a peculiar appearance of skin below left nostril, the face growing thinner below left eye and above left angle of mouth. Examination showed distinct atrophy of middle and lower third of left side of face. Every few seconds clonic and then tonic contractions of temporal and masseter muscles occur. There is also atrophy of left half of tongue and left floor of mouth. On the upper left lip is a pigmental "scar." Pain sense is slightly diminished on the left side, and thermometrical measurements of external ear show a difference of one degree in favor of the right. The only autopsy obtained in a case of this disease (Mendel's) would tend to show that it may be due to a peripheral neuritis, and that the diseased parts, the descending root of the fifth nerve and the substantia ferruginea, have trophic functions, presumably connected with trophic fibres in the peripheral branches of the trigeminus. It often begins after some acute infection—erysipelas, or follows facial traumatism. ("Medical Record," March 15, 1890). A. F.

DISLOCATION OF CERVICAL VERTEBRÆ WITHOUT FATAL RESULTS.

G. L. Walton, M.D. ("Boston Med. and Surg. Journal," May 8, 1890). The writer calls attention to the fact that these cases are not rare, though probably sometimes overlooked through lack of familiarity with the diagnostic

features. This is true more particularly of the unilateral form, in which the cord may escape pressure. Such cases present themselves generally with a history of a fall, possibly followed by temporary paralysis of the legs and arms, with or without retention of urine, but showing at present either no paralytic symptoms or symptoms comparatively limited. Lack of mobility will be noted, passive motion being limited in certain (if not in all) directions, and such efforts cause pain. More or less sensitiveness is present, and perhaps a prominence of transverse processes on one side. In typical unilateral dislocation the position of the head resembles that of torticollis; but the cervical muscles on the side which would produce the deformity in torticollis are lax, while those on the other side are more or less tense, through being put upon the stretch. He calls special attention to the view from the back, one ear being higher than the other, the head somewhat rotated, and in some cases set off as a whole to one side.

In addition to the five cases previously reported by the writer in *THE JOURNAL OF NERVOUS AND MENTAL DISEASE* and in the "*Boston Medical and Surgical Journal*," three more are given.

The first is that of a carpenter, who fell fifteen feet from a staging and was taken up insensible. The only symptoms following were pain in the abdomen, thorax, back, shoulders, and neck. Recovery followed, excepting that shooting pains persisted in the back of the neck. The knee-jerk is still active, and a trace of clonus exists on the left. Motions of the head are limited as to rotation, backward flexion and tilting. The position is characteristic of unilateral dislocation, the chin pointing toward the left shoulder, the muscles on the right being lax.

The second case is that of a waiter, who was thrown from a car, striking the vertex. He was unconscious a short time. Retention followed. For two weeks complete loss of motion in arms and legs, with loss of sensation to the groin. Gradual improvement. Extensive scars of the scalp. Movements of the neck limited in all directions. The head held well forward and somewhat to the left. Forced movements cause pain. Tenderness in lower cervical region. Gait stiff and unsteady; knee-jerk normal. Sensation normal, except on the ulnar side of both arms, including the ring and little fingers. Atrophic paralysis with degenerative reaction in extensors and intrinsic muscles of the hand on the left. Quite unaffected are the biceps, triceps, and deltoid, the flexors, pronator radii teres, and supinators.

The writer discusses spinal localization as studied by Ferr n and Yeo, Herringham, and Ross and Thorburn, recalling the case of a boy, published in the previous paper, in whom unilateral dislocation of the third upon the fourth produced atrophic paralysis of the left arm, commencing as high as the supra- and infra-spinatus muscles, while the location of anæsthesia, in the case just reported, would indicate disturbance as low down as the seventh cervical nerve-roots.

The third case was that of a currier, who fell fifteen feet, landing in the sand on the vertex. There was loss of power in the legs, without urinary disturbance. The chin was immovably fixed on the chin for some time. There was stinging pain in the back, and grating in the neck, with pain on movement, with numb sensation in the back of the head. The symptoms have disappeared, excepting that the head is still held somewhat stiffly forward, and there is limitation on backward flexion.

Society Reports.

AMERICAN NEUROLOGICAL ASSOCIATION.

*Sixteenth Annual Meeting, held at Philadelphia, June 4th,
5th, and 6th, 1890.*

Dr. E. C. SPITZKA, President; Dr. G. M. HAMMOND, Sec'y.

PRESIDENT'S ADDRESS.

IN welcoming the members of the American Neurological Association, on the occasion of their Sixteenth Reunion, it is a pleasant double duty to acknowledge the hospitable courtesies of the local neurological body, and to congratulate you on your choice of Philadelphia as the place of meeting. In the highest sense of the word, the city of Rush, Ray, Kirkbride, Mitchell, Morehouse and Keen, as a centre of achievement and research, occupies a proud place among the neurological foci of the world. It was here that he whose name proudly represents our profession on that historical Declaration which was drawn up in the hall but half a mile distant from this one, anticipated that principle of treatment of the insane, which Chiaruggi, Pinel, Conolly and Tuke perfected and carried into a larger and better prepared field of practice. In a more

recent and equally critical period of our history, it was here that the wounded collected from the battle-fields of a hundred internecine combats furnished material for the novel and marvelous studies on reflex nervous symptoms following gun-shot wounds, which have been confirmed by military surgeons every subsequent war, albeit a physiological explanation is still a desideratum.

Perhaps no other special branch of medicine calls as emphatically as neurology does, for a renewal of that union of biological and strictly clinical studies, which had been nearly, if not altogether broken during the past three decades. Here and there earnest students with ideals higher than those of the mere bread-winner, kept alive the tradition that biology owes its chief impulse to medicine, and perhaps unconsciously kept open a channel of communication, which seems destined to bear the return current by which the former science may repay a hundred-fold the loan made by the old masters of our art.

No centre of learning on this side of the Atlantic can assume to itself as justly as Philadelphia can, the claim to having maintained alive the pure flame of biological, that is, strictly scientific medicine. Need I mention a Leidy or Harrison Allen in the general field? Need I refer to the fact that notwithstanding the vast opportunities in this direction enjoyed by other lands, it was left to Philadelphia's physicians¹ to furnish the first reliable descriptions of the mongolian brain?

It is with a pride which I trust may be deemed excusable that I venture to point to the prospect of enjoying at this reunion, a continuance of valuable neurological contributions in the speculative as well as the practical psychological, the biological as well as the clinical fields. We are to receive a report on the anatomical findings in the historical first case of athetosis, by the son of the discoverer of that motor disturbance. An almost unprecedented case of spinal surgery is to be presented to us to-morrow by one of our Philadelphia confreres. With other interesting com-

¹ Dr. F. X. Dercum, see Proceedings of Fifteenth Meeting, JOURNAL OF NERVOUS AND MENTAL DISEASE, 1889.

parative anatomical and pathological specimens, the brains of scientists, those of criminals, and those of anthropoid apes will be exhibited. Above all, we are to be favored by one whose name is a household word in neurology, with a paper on a subject already announced, and two additional communications of such interest and originality, that you will gladly forego a lengthy inaugural on my part, and permit me to yield my place on the programme to him. I am aware that your presiding officer is expected to present his neurological work of the year in the form of an address. But on this occasion, the number of papers is so great, and their prospective interest so high, that I will beg the privilege of reading my own papers by title² in order that you may listen to the one whose name fitly leads on our programme. I ask your attention to a paper on "Spinal Chorea," by one who needs no other introduction than the mention of the name—S. Weir Mitchell.

DR. S. WEIR MITCHELL, read a paper on "Unusual Cases of Chorea, possibly involving the Spinal Cord." (See page 427)

DISCUSSION.

Dr. C. L. DANA said he thought the view taken by Dr. Mitchell, as to the relation of heredity in chorea, was the correct one.

Dr. B. SACHS suggested that some of the features Dr. Mitchell had mentioned, in detailing his cases of chorea, resembled somewhat the peculiar disorders of movement noticed in children with spastic palsies. Some of these

² 1. Experimental Results (Hemi-Contracture in Extension and Spinal Chorea), Following Partial Unilateral Section of a Dog's Cord at the Level of the Foramen Magnum. 2. On a Tract Related to the Crossed Pyramidal Tract Demonstrated by the Atrophy Method to be Connected with the Thalamic and Sub-Thalamic Regions. 3. On the Frequent Coincidence of Diabetes in the Parent, with Mental Defect (Abulia, Adolescent and Pubescent Types of Insanity) in the Offspring. 4. The Otter's Brain: A Link Connecting the Brains of Land with Those of Aquatic Carnivores. 5. The Spinal Nuclear Anatomy of the Marine Mammals. 6. The Same in the Hippopotamus and Manatee. 7. The Same in the Chimpanzee. 8. The Nuclei of the Thenar Muscles Demonstrated by Their Absence in the thumbless Ateles, and Relative Hypertrophy in Man as Compared with Apes. 9. Influence of Exudative Meningitis on Cord-Nutrition, as Illustrated in Spina Bifida.

conditions made their appearance at a late date, and might be either severe or mild in character. He did not of course oppose Dr. Mitchell's theory, and really believed it to be the correct one.

Dr. MITCHELL said the view he had advanced was rather an hypothesis than a theory.

Dr. SACHS gave as his reason for introducing the idea, the fact that he had seen these peculiar disorders present after every trace of the early paralysis had disappeared. He approached this subject cautiously, but inasmuch as in two of the cases cited the patients had shown exaggerated reflexes, which were also found in the congenital case, it was possible that one or other of the cases might be congenital palsy, in which the symptoms of palsy had disappeared. He could not abandon the idea that some of the cases described might have shown changes in some part of the brain. He should not be surprised if we still must add to our causes of chorea.

Dr. E. D. FISHER stated that he had seen a great many cases such as Dr. Sachs had referred to. In the almshouse they had both adults and children whose history was that of congenital paraplegia, or hemiplegia of cerebral origin, and in which the paralyses were associated with well-marked choreic movements. The effect of strychnia would not preclude the idea that the movements were of spinal origin. The question might be raised, perhaps, as to whether the primary lesion had been spinal or cerebral. We certainly, in these cases of cerebral origin, found a secondary degeneration. It had seemed to the speaker that many of these cases, where the condition had remained chronic for so long a time, were really not of the nature of ordinary chorea, which was a self-limited disease. He had always regarded these conditions as associated with sclerosis, leading to degeneration in the nerve-cells and nerve-fibres and to descending degeneration in the cord. He had looked upon this as cerebral in origin because it was found that the mental powers, though not greatly affected, showed some dullness, especially in the acquisition of knowledge.

Dr. F. X. DERCUM thought it was not at all improbable that a spinal centre might be affected. A few days since a case had been brought into the hospital similar to that mentioned by Dr. Mitchell. The boy was put to bed, and was soon much better. When admitted, however, the movements of the arm and leg were very marked, and different in character from those of ordinary chorea, and

would lead to the conclusion that the condition was like that found in the dog, which was known to be of spinal origin. No doubt, there existed a form of chorea which was purely spinal.

Dr. MITCHELL said that many years ago he had stated his belief that ordinary chorea should be classified into three groups. It was true that cerebral changes could arise in these choreic conditions, but it was rare to see these troubles affect both sides, no matter what the changes were in the brain. It was especially rare to see the condition in both lower extremities. Therefore the first case cited was not chorea, resulting originally from paralysis, accompanied by descending degeneration of the cord.

DOUBLE CONSCIOUSNESS.

Dr. MITCHELL then alluded to the notorious case of Ansell Brown, who had left his home, assumed another name, and, as asserted, had lived for some time without knowledge of his previous existence. On regaining control of his proper identity he had returned home. Hypnotism had been recently tried on him, and while under its influence the man's mind could be made to revert to incidents in his fictitious existence, while of his real identity he would then know nothing.

Dr. C. K. MILLS had seen a number of epileptic cases in which there had existed in the patient a change of ideas as to personality, but of very few cases in which the intervals were of such long duration as in the case referred to by Dr. Mitchell. The speaker had a patient who at times was in a similar condition. This man had left his home, and had started with the intention of going to Atlantic City, and had found himself, after two weeks, in Lynn, Mass. This was, however, a traumatic case, the patient having suffered a fall from his horse—the train of mental phenomena coming on after the injury.

Dr. R. L. PARSONS thought the subject was of great importance from a medico-legal standpoint, as the condition of changed consciousness might be urged in extenuation for the commission of crime.

Dr. C. L. DANA said that some years ago, he had occasion to go over the literature of the subject. He was then able to find some 20 or 30 cases identical in character with that of Dr. Mitchell's. All the best cases, however, were so old that it was very gratifying to find one of recent date.

The trouble was very much allied to epilepsy, and the result of his study of the subject was to lead him to think that there were graduations of the disease, and that many of these cases were epileptic.

Dr. P. C. KNAPP cited a case of hystero-epilepsy, in a woman. This patient had suffered from hysterical fever and various other nervous phenomena of a pronounced type. Besides periods of coma, she had spells of changed consciousness, waking up with the idea that she was in some remote place. She would address the physicians and nurses as "uncle" and "aunt." As to the question of spinal chorea, he was struck with the analogy of Sydenham's chorea with the conditions of ataxia, tonic spasms, and associated movements, of which he had recorded several cases two years ago, and to which some of these cases of chorea bore a striking resemblance. It did not seem that we were yet in a position to assume any one of these motor disturbances as having any focal significance. They might have their origin anywhere within the motor tract, and he hoped to show that tumors were not infrequently the cause.

THE WEATHER IN RELATION TO NEURALGIC PAIN.

Dr. WEIR MITCHELL stated that for many years, a patient of his, a retired army officer, and now professor of physics, had, at the speaker's suggestion, made elaborate studies and observations of the effect of variations of the weather upon neuralgic paroxysms. The patient was a constant sufferer from neuralgic seizures, and the results of his scientific findings had lately come into the possession of the speaker. The charts were so elaborate and the written material so voluminous, that there had been no opportunity for even segregation of the salient points. Briefly it might be stated that the maximum of pain bore direct proportion to the prevalence of storms, and that the aurora was a certain precursor of neuralgic excitation.

Dr. G. L. WALTON then read a paper entitled "A Contribution to the Study of Traumatic Neuro-Psychoses." (See page 432.)

THE PRESIDENT regretted that the works dealing with this subject were open to the stigma of having been written for trade purposes. It was important in discussing the subject to avoid anything but its clinical aspect.

Dr. P. C. KNAPP could not agree with the author of the paper in the position he had taken. Page had deliberately

neglected some of the most important symptoms in neurology, and his work could only be considered that of a special pleader. He had practically ignored the subject of reflexes, knee-jerk, and electrical reaction. Such a work was one upon which they, as neurologists, could place no value. The theory of Charcot, that these motor phases were hysterical, was pretty well exploded. Looking over his own notes, he had failed to find more than one case of typical hysteria resulting from injury. Many of the cases cited, presented symptoms of a neurasthenic state, but just how far such conditions were functional or organic, was uncertain. Several cases were then detailed, which, the speaker thought, bore out the conditions indicated by Oppenheim as traumatic neuroses. In one patient, since dead, whose case had been the subject of medico-legal investigation, the condition had been such that examination had been almost impossible, although the patient used morphine freely. There had been general complaint of headache and backache, vomiting, palpitation, a weak and rapid pulse, tenderness over the posterior nerves, and a good deal of paresis, with loss of knee-jerk. The pupil reaction could not be obtained, though whether this was due to the morphine or the disease the speaker could not say. Another case was that of an officer in the navy, who had dated his trouble to falling out of the berth of a sleeping-car. Here there had been no question of litigation. He had drifted about the country seeing different physicians. The diagnosis was disseminated sclerosis. This man had died in three or four months. No autopsy had been made, but the speaker had thought that the case went to show that in a certain proportion of cases there existed organic disease. He agreed that there were many symptoms which were subjective and which might be imitated, such as diminished sensibility, tenderness, facial expression, headache and exaggerated reflexes. But there were many minor symptoms partly subjective and partly objective; for instance, limited anæsthesia. It was a question whether it was quite possible for a malingerer to make the distinction with the requisite definiteness. The question of tenderness might be tested by the finger on the patient's pulse, as any marked painful sensations would be followed by increased rapidity of the heart-throbs.

Dr. J. J. PUTNAM thought there were but few persons who could carry out successfully a system of deception during a long and searching examination, much less through two such investigations.

Dr. F. X. DERCUM said that there was a union of two factors. In some cases there existed actual cause for painful back. It was remarkable how the same story would be repeated unsolicited. It required a person well trained in the subject to relate such symptoms. Where there would be diminution of the heart-beat, great awkwardness of movement, marked disorders of sensation, errors in locating points of contact, and many other mistakes which a healthy man could not make. Many of the symptoms were such as the patients could hardly pick out for themselves. Pain, referred to the back was of two kinds, neurasthenic pain, and pain on superficial pressure. In every case there was pain on deep pressure, and patients never forgot the area in which this pain existed. It was immaterial whether the initial trouble was actual organic disease or profound functional disturbance, the fact remained that these people did not recover.

Dr. W. R. BIRDSALL agreed that it was the more conservative view to say that there was possibly a traumatic lesion in a certain number of these cases, still he thought the great majority showed functional phases. The chief element in the production of these conditions, even if there existed organic changes in addition to the functional derangement, was emotion and psychic shock. Nearly all the cases which he had seen had been cases which presented functional disturbances, the patients acquiring as a result of fear of the consequences of the accident, a habit of studying their own symptoms. The importance of muscular strains and bruises were magnified in the mind of the patients day by day, until they became psychical troubles instead of local. The phenomena following shock by the electric wires were of a similar character.

Dr. D. INGLIS did not agree with the author of the paper. The profession had to take one position or the other in deciding whether these cases were organic or functional, and then a jury might do as it thought fit. It was a fact that after lingering along, the patient, when a legal decision was reached, rapidly began to recover. Such a patient might not intentionally simulate. A man after injury to his back and in which actual pain was present, might honestly feel and plead inability to move. As long as his case remained *sub judice* this condition might obtain, but when the case was settled he began to move about and work, and his pain disappeared. In these cases, were

increase of cutaneous sensibility urged, they would also claim the same to the electrical current. It was a good plan in such cases to connect four or five electrodes with the switches, and by fastening these all in position with one of them on the alleged sensitive area, the patient would not know at what point the current was going to enter, and when it did enter at the point claimed as hypersensitive, he would flinch uncontrollably.

Dr. C. K. MILLS thought there were at least three classes of cases resulting from injury: (1) Pure fright; (2) cases in which the indications were clear that fracture or hemorrhage, or other serious lesion had taken place; (3) cases in which the symptoms presented were both objective and subjective, with a preponderance of the latter. As to the third class, the true explanation seemed to be that there was a real lesion, giving rise to a certain set of symptoms forming a groundwork upon which the patients erected for themselves an enormous psychical superstructure. He thought that the existence of some organic lesion, whether myelitis or the result of hemorrhagic pressure upon some delicate part of the nervous system, would explain many of the symptoms peculiar to this class. He had seen patients presenting the same phenomena for months, which it would have been practically impossible that they could have repeatedly enacted upon the same lines. To assume it was an unscientific argument. Fear he did not think could produce the symptoms. Suggestion might do much in certain cases, but in most there were elements which precluded the idea of continuous suggestion.

Miscellany.

QUADRUPLETS

J. de Leon, M.D., of Ingersoll, Texas, writes to the "Dietetic Gazette" of his experience in delivering a woman of four living children, and gives the credit of successfully raising them to Reed & Carnrick's Infant Food.

The doctor long since has found this Food possessed qualities that he failed to find in the others.

The case is rare of raising quadruplets; they most always are puny and die very early.

This undoubtedly speaks well for the merits of the Food.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

A FORM OF POLY-NEURITIS, PROBABLY ANAL-
AGOUS TO OR IDENTICAL WITH BERI-BERI,
OCCURRING IN SEA-FARING MEN IN
NORTHERN LATITUDES.¹

BY JAMES J. PUTNAM, M.D., OF BOSTON.

ON the 21st of last October a man, thirty-nine years old, the mate of a fishing-vessel that had just arrived from the Grand Banks, was referred to me by my friend, Dr. M. A. Morris, of Charlestown, and gave the following account of his condition:

He was suffering from weakness in both arms and legs to such an extent that he could walk only with difficulty. His legs felt numb and prickly, and he had observed an actual loss of sensibility of the skin. This feeling of numbness extended up the legs and thighs and over the lower part of abdomen to the umbilicus. The arms were in a similar condition, but to a less degree. No affection of the face or eyes had been noticed. There was no pain anywhere, and no affection of the sphincters of the bladder or rectum. He said the legs were swelled, but not so much so as they had been a short time before; the calves and the inner surface of the thighs felt sore and lame.

¹ Read at the Annual Meeting of the American Neurological Association in Philadelphia, June, 1890.

He reported that he had been well up to two weeks previously. The first symptom noticed was pain on the inner surface of the thighs, on the 7th of October. The next day he felt sick, and the legs and thighs were swelled. He kept in his bunk for several days, feeling weak and depressed, with a steady diminution of strength in legs and feet. After this he had improved only slightly. He had had no fever, so far as he knew.

On examination the patient was found to be pale, with a yellowish cast to the face. His gait was waddling, the legs being used like stumps, and the feet brought down flat on the ground, as if simply hinged at the ankle. He had considerable difficulty in getting his coat off and on. In going down-stairs he was obliged to turn sideways and to bring both feet down upon each step. There was slight toe-drop, and marked swaying of the body when the patient stood with the eyes shut. There was no inco-ordination in the arms or hands. The grasp by the hand was feeble on both sides, more so on the left. Extension of the fingers and carpus was imperfect on both sides. The sensibility of the hand to touch was so nearly perfect that the patient could feel the lightest contact with my finger everywhere. The sensibility to contact in the feet and legs was but little below the average, but the character of the sensation imparted was not quite normal. The prick of a pin was promptly and distinctly felt, and, indeed, the patient's answers suggested some degree of hyperæsthesia. A very light touch was also felt over the abdomen below the umbilicus, but, as compared with the area above the umbilicus, the sensibility of the affected portion was slightly impaired, or, at least, the contact gave rise to a feeling of a different kind in the two parts. A piece of metal of the temperature of the room was distinctly felt as cold whenever tested. The knee-jerk was absent on both sides, even during "re-enforcement."

The calves of both legs were large and tender, distinctly more so than normal, by the patient's account, and there was pitting on pressure along the shins. The muscles over the inner surface of the thighs were also

tender to deep pressure, and to a slight degree the muscular masses of the arms. No enlargement of the spleen could be made out. Inquiry into the patient's previous history showed that he had been in all respects a temperate man. The pulse was 78, full and strong; temperature, 99° F. in the mouth.

To revert to the surroundings of the patient before the attack came on, it appeared that for nearly six months before his sickness he had been on a fishing-vessel off the Grand Banks. The vessel had been insufficiently provisioned, and the captain had insisted on protracting the trip, so that for some time the crew had had little else to eat except molasses, fried pork, and pan-cakes, and the water had been foul. Nine or ten other men besides the patient had been affected like himself, and most of them more seriously. The first one was attacked two months before he was, namely, in August. Their legs were badly swelled, so that the pitting on pressure was frequently noticed.

I learned, further, that some of these men had been treated at the Marine Hospital, and I was enabled, shortly afterward, through the kindness of the surgeon in charge, Dr. Fairfax Irwin, to learn something of their history and to examine the one or two that remained. Dr. Irwin reported to me that, on account of the œdema, the malnutrition, and, in one or more of the cases, the typical purpuric condition, he had made the diagnosis of scurvy; and it certainly seems highly probable that this affection accounted for at least a part of the symptoms.

Of the two patients remaining in the hospital, one was a typical example of multiple neuritis, presenting well-marked wrist-drop and toe-drop, impairment of sensibility of the hands and feet, and more or less tenderness on deep pressure into the muscular masses of the arms and legs. He had been improving, and soon afterward left the hospital.

Some months after these cases were brought to my notice, a patient presented himself at the Massachusetts General Hospital, presenting the symptoms of multiple

neuritis in even a more severe form than either of the cases already described, and bringing a note from his physician, Dr. Stone, of Wellfleet, saying that he had seen several other cases of the kind. The patient was a seaman, twenty-one years old, single, in good circumstances, and with absolutely no history of constitutional disease. He had been well up to the 1st of July, 1889. At that time he was in a fishing-vessel off Block Island, near Newport, R. I., having left his home in Wellfleet early in June. The vessel was well-provisioned. The first symptom consisted in a sense of numbness in the feet, which began at the toes and rapidly spread upward, but never reached more than midway up the thigh. Soon afterward the fingers also began to be numb, and the disturbance extended up the arms. At the time of the illness he was feeling as strong and healthy as ever in his life.

The weakness of the arms and legs began at the same time with the numbness. Soon after the hands had begun to be affected—that is, the 26th of August—the patient began to suffer severely from dysentery of a serious character, which lasted for two weeks, and was followed by a severe diarrhœa, which continued for three months. He remained in bed all the time, having a very large number of movements daily, and indeed his legs became so weak that when he tried to stand he found it impossible to do so.

In July, soon after the symptoms began, the legs, as well as the lower half of the thighs, began to swell, and became highly œdematous, pitting deeply on pressure. At the same time he suffered from severe pain, mainly along the shin, accompanied by marked soreness on pressure. It is possible that this pain was due to the swelling, since the feet were not painful. There was no pain in the arms. During a great part of his illness he was on shore, having left the ship after having been sick for a week or two. During the height of his sickness he had some attacks of unconsciousness, lasting for an hour or so, accompanied by high fever, which was thought to be of malarial origin. For two or three weeks, just after the dysenteric attack, he was unable to control the sphincter of the bladder. At the time

I saw him he had improved considerably, both as regards the paralysis, which had been so great that all motion of the toes and feet was abolished, and the muscular wasting, which had been very marked.

The patient reported further that all the men aboard the ship, eight in number, were affected like himself to a greater or less degree, except that only one had the dysenteric symptoms.

The paralysis was about equally great with them all, but did not, in most cases, last so long as with him. The other member of the crew who had dysentery was attacked with it at about the same time with himself, and afterward died.

The physical examination of the patient showed his condition to be typical of the so-called multiple neuritis, if not, indeed, something worse. No motion was absolutely impossible, but a high degree of toe-drop was present and the extensors of the carpus and fingers were much affected. He walked slowly and with the aid of a cane.

The examination of the sensibility showed that the sense of contact on the foot was slightly blunted to a little above the ankle. He was a good deal at a loss to localize the sensation, mistaking the sole of the foot for the dorsum, etc. The outer side and back of the foot seemed to be more affected than the sole. There was considerable delay in conduction. A piece of metal of the temperature of the room was sharply felt, even on the great toe. His sense of position seemed slightly impaired, so that he had difficulty in standing with the eyes closed, and he failed to state correctly the position of the toes. The foot felt cold to the touch. There was no static ataxia of the hand, and the sense of contact was apparently normal for the tips of the fingers. Two points were distinguished at about 1 mm. The electrical reactions for the muscles of the legs were much diminished. (*Vastus internus* and *rectus femoris*; F. reaction, 6.5 (normal, 11-12). *Sartorius* reaction almost normal. All the muscles below the knee reacted to F. at about the same strength of current as above. G. reactions; no local contractions from weak or moderate currents.

Currents of 20 m.a. caused rather feeble reactions everywhere, $->+$. The peritoneal nerve reacted to 5 m.a. at first, the tib. anticus responding; but, after one or two closures, the contractions died away and could not be obtained.)

In October, 1881, Dr. F. C. Shattuck reported in the "Boston Medical and Surgical Journal" a series of cases evidently identical with those to which I have referred and occurring under precisely similar conditions.

The patients were fifteen in number, and embraced all but four of the officers and crew of a fishing-vessel, the *Nellie Swift*, that sailed from Provincetown for the Grand Banks on June 5, 1880. The first patient fell sick on August 1st, complaining of soreness in the calves of the legs and a dead feeling in his ankles, which was soon followed by swelling in those parts. The subsequent symptoms in this and the other cases consisted in extensive and severe anasarca, pain and numbness. One of the patients died, having presented the above symptoms and, in addition dyspnœa, with effusion in the chest. The urine also had been scanty and high-colored, free from albumen, but containing a few granular casts. An account of the autopsy was sent to Dr. Shattuck by Dr. J. M. Crocker, of Provincetown, under whose care the patient had been. Serum had been found in the pleura, pericardial and peritoneal cavities. The heart was flabby, but otherwise normal. The kidneys were normal in appearance and size. The spleen was unusually small. Dr. Crocker further reports that none of the cases had, so far as he knew, presented hemorrhages or other symptoms of scurvy beyond œdema of the ankles; and, further, that the drinking-water which was used on the ship was thick and ropy.

As has been stated, the vessel was off the Grand Banks when the sickness broke out. During the previous winter, however, it had been engaged in the fruit trade in the West Indies, visiting Nassau among other places. The provisioning of the vessel had been practically the same as that of other vessels in the same trade. In a letter written a few months later, Dr. Shattuck also referred to the epidemic

which broke out on a Brazilian man of war, said to have been improperly provisioned and in an unhygienic condition. Leaving Brazil, the ship sailed for Europe, touched at points in the Spanish Peninsula, and passed through the Suez Canal to Aden, where the disease, which was manifestly of the same character with that of the cases already reported, first broke out. The cases were treated in the Marine Hospital in San Francisco, and an account of them was published in the hospital reports. Dr. Shattuck concluded that the cases must be of the nature of Beri-Beri, and quotes Dr. Roosevelt as having described a series of cases which occurred on the *Henry S. Sanford*, which sailed from Hong Kong for New York, July 20, 1886. Of a crew of eighteen, twelve were attacked, and several cases were fatal. The outbreak of these epidemics on the last two vessels named is perhaps remarkable from the fact that it occurred so long after the vessel had left the Southern ports where Beri-Beri is to some extent endemic. In one case the interval was three months, and in the other six months. I have a number of references to other cases where the same fact was observed. In the two sets of cases to which I have referred, even this explanation is wanting, and we are obliged to fall back on the conclusion that some infectious form of multiple neuritis occurs in northern latitudes, a view supported by the occasional occurrence of such cases as that described by Rosenheim and published in Vol. XVII. of the "*Archiv. f. Psychiatrie*," and the one reported by myself at the meeting of this society in 1888. Both were severe cases of acute generalized neuritis, running a rapid and fatal course, but without œdema.

Wishing to learn whether other cases of this sort had been observed by physicians in the seaboard towns, I sent out a number of circulars, and obtained fifteen answers, of which those from six physicians—Drs. Wm. S. Birge, of Provincetown; S. F. Quimby, of Gloucester; G. B. Stevens, now of Roxbury; S. T. Davis, of Orleans; Benj. D. Gifford, of Chatham; W. N. Stone, of Wellfleet; and E. E. Hawes, of Hyannis—are of decided interest.

Dr. Birge reported having seen seven cases, five from one

vessel and two from another, belonging to a fishing-fleet which had returned from the Grand Banks. Two of the cases had proved fatal. The symptoms described were evidently identical with those of the cases reported by Dr. Shattuck and myself. One of the vessels may have been the same with that from which my first patient had come.

Dr. Quimby had seen the master of a fishing-schooner who had these characteristic symptoms, pain, and paralysis in the lower extremities being very marked. The symptoms had come on, as he thought, as a sequel to the influenza of the past winter, but as I have been looking in vain for cases of neuritis following the influenza, I think this explanation is not probable.

Dr. Stevens called my attention to a case which he had reported in the "Boston Medical and Surgical Journal," June 16, 1887, occurring in the mate of the barque Charles G. Rice, which arrived in Boston, May 12th, of the same year, from Manilla, after a passage of one hundred and twenty days. The symptoms in this case were essentially diarrhœa, œdema, and, eventually, numbness of the legs without paralysis. The point of particular interest in this case is that the patient was well when he left the port of Manilla and became ill about a month after that time.

Dr. Davis had seen "a lot of cases where men who had handled fish complained of numbness and great swelling of the hands and great difficulty in using the extensors." He had considered these due to the irritating action of the fish on the hands. In view of the uniformity of employment of all the men, this explanation should receive full weight; but the fact that a much larger number of cases seems, as far as we know, to have occurred in certain years than in others, it would seem that neither this nor any other local cause could be the only one at work.

Dr. Gifford had seen one patient, a sea-captain, with symptoms that might perhaps be of this character.

Dr. Stone has sent me the notes of five cases, of which at least three may have been of this class. The diagnosis is, however, not certain, and in some of the cases other well-known causes of general neuritis were present.

The first of these patients was a man of forty years; a seaman, of temperate habits, and free from constitutional diseases, so far as is known. The symptoms had consisted in progressive numbness of the legs reaching to the knees, and toe-drop, the whole increasing to its maximum in a few weeks. He improved to a certain point, but never entirely regained the use of the extensors of the foot. This case occurred two years ago.

The next patient was a man of forty-five years, who had followed the sea until the last five years, since when his occupation has been that of a sailmaker, his place of business being on the beach, where he was exposed to wet and damp. Three years ago he was attacked with what appeared to be sciatica of one leg, soon extending to the other, and lasting for a number of months. From this he recovered, but a year ago it returned and remained for some three months. Three weeks ago he was again attacked with severe pain in the limbs and feet, the hands becoming badly swollen. The swelling subsided after a time in the hands, but attacked the feet, and at present is spread over the whole length of both legs. The pain is still very severe.

The next case is that of a man of thirty-five; of previously good health, temperate habits, and free from constitutional diseases; he had always followed the sea. His symptoms had consisted in an acute onset of pain in both legs and in general in the distribution of the sciatic nerve. This pain was worse in the afternoon and evening, as a rule. It subsided in the course of a week or two, but for three months more his muscles were so weak he was unable to walk even with crutches. Four or five months later he could go about with difficulty, and before the end of a year he was able to go to sea again, though still, at the end of nine years, showing some awkwardness in his gait, and using the feet as if they were of wood.

The fourth case was a man of sixty-three. It was a typical case of multiple neuritis, but is not reported here at greater length because the habits of the patient suggested that alcohol may have been the cause of his sickness.

The fifth case was that of a man thirty-five years old ; always in good health, and a member of the life-saving service crew, his duty being to patrol the beach at night. The beach, when not frozen, was sandy, making the walking difficult. A year ago he came to Dr. Stone complaining that while walking his toes felt as if there was sand in his shoes, and that this feeling had increased of late. He had noticed that on going down a sharp decline the knees were apt to give way, so that he would fall. Of late the numbness had extended to the calves of the legs, and he had recently had pain through the abdomen to the back, and down the thighs.

Dr. Stone was kind enough to give me these notes from his memory, but I thought it better to put them in on account of the obscurity concerning the etiology of this class of cases, thinking they might throw some light upon it.

Dr. Hawes, of Hyannis, says: "About seven months ago I had a fisherman (I am hospital surgeon of this port) come to me with an ulcer on his leg. I treated 'it, and he went again to his vessel. He said: 'A few weeks ago I was taken lame, hardly able to stand erect, and I feared I should be paralyzed.' It seems, as he rallied from that, the sore formed. I judged it might be rheumatism, and so can say no more about it. But since receiving your letter I have thought it might be the Beri-Beri that the man tried to explain, and that the sore might have been an ulcer following the above-named trouble."

To sum up, it would seem that in 1881 and in 1889 there were epidemics of this infectious disease, whatever name it may deserve, occurring among the crews of vessels fishing along our northern shores, and that sporadic cases of the kind have occurred during the interval.

The group of cases that I have seen or heard of amounts to fifteen or twenty, exclusive of Dr. Shattuck's fifteen, and exclusive of those reported in answer to the circular, the number of vessels being five, three of them from the Grand Banks and one from off Block Island.

I have received a letter, within a few days, from one

patient, whose case I have reported, saying that he still suffers from numbness of the feet and toe-drop, though it is now about a year since he was first attacked, and saying further that another of the crew is still quite sick. It is possible that, either primarily or secondarily, the spinal cord was affected as well as the peripheral nerves. He further notes that his vessel was a mackerel fisherman, while those off the Grand Banks were cod fishermen. The possibility of infection from the fish should be considered, and the letter from Dr. Davis is interesting in this connection. He also says that the vessel on which he and his crew were employed had not at any time been engaged in the Southern trade, so that this possible cause of infection, which was present for Dr. Shattuck's cases, was not present here.

REMOVAL OF THE HAND-CENTRE FROM THE CORTEX
CEREBRI IN A CASE OF FOCAL EPILEPSY.

In the "Medical News," April 12, 1890, W. W. Keene, M.D. reports the case of a boy, aged six years, who fell from a hay-mow upon a plank flooring when 14 months old. There was no evidence of head injury, except temporary unconsciousness, followed by extreme irritability, which has existed up to the present time. At the age of two and a half years he became epileptic, about 80 per cent. of the attacks beginning in the right hand. It was also observed that a large dose of bromide was almost always followed by paralysis of the right hand. When the attacks began his vocabulary included about 40 words, but this has gradually diminished to three. Operation being decided upon, the hand-centre was exposed and located by means of the battery, but nothing abnormal was found here. All the centre for the hand and wrist was removed. Speedy recovery took place, and although he has had a few attacks of *petit mal*, there have been no severe convulsions. His vocabulary has increased 20 words, and he is more docile than formerly.

L. F. B.

TUMOR OF THE THALAMUS, MORE ESPECIALLY OF THE PULVINAR, PRESENTING WERNICKE'S PUPIL REACTION.¹

By F. X. DERCUM, M.D.,

Instructor in Nervous Diseases, University of Pennsylvania.

OPTHALMOSCOPIC EXAMINATION BY CHAS. A. OLIVER, M.D.

H. N., male, aged thirty-nine years, of German descent, and a bridge and wharf builder by occupation, was first seen on February 3, 1890, in consultation with Dr. A. S. Gerhard. He had never had any previous illness of moment. He had never abused either alcohol or tobacco, but admitted great sexual excess.

Two years ago he began to suffer from attacks of vertigo, and a little later from headaches, the latter being now and then accompanied by temporary blindness. The headaches were not constant, and occurred at irregular intervals. Nothing new was noticed by him until April, 1889, when his right leg began to feel stiff and numb, and he occasionally struck the toes of his right foot against the ground in walking. This condition persisted until the following August, when in addition curious spells, in which his right arm was drawn up, began to make their appearance. These spells at first occurred at intervals of several days, but gradually grew more and more frequent, until at present they occur every few minutes. Of late the headache and vertigo have not been marked, and no new symptoms have made their appearance. There has been, however, some weakness of the right arm and leg.

The present condition was now noted. The man was exceedingly well-built and healthy looking. He was of good frame and has fine large muscles. When asked to walk, the right leg dragged a little and was slightly spastic. The grip of the right hand was weak as compared with the

¹ Read at the Annual Meeting of the American Neurological Association in Philadelphia, June, 1890.

left. The knee-jerk on the right side was distinctly exaggerated, while it was normal on the left. Tested by the æsthesiometer the numbness complained of in the right arm and leg proved to be largely subjective though the points were not separated quite as readily as upon the left side. The muscular sense of the right side, however, proved to be much impaired as was also the sense of pressure. He was tested as follows: The patient being blindfolded, two cups exactly equal in shape and size were taken. One was partly filled with water, the other remaining empty. When these cups were placed in turn upon the partially extended palm of the left hand, he instantly distinguished the full from the empty cup. His replies were invariably correct. Not so, however, when the experiment was made with the right hand. Here the answers were without exception erroneous, the patient not even making a single fortunate guess. He was next seated at a table, still blindfolded, with the hands resting upon the table, palms upward. Here the experiments were repeated, with exactly the same results as before.

The temperature sense was now tested by means of two spoons equal in size and shape, the bowl of one being warmed over a lamp and the bowl of the other chilled by means of ice-water. Both were applied, thoroughly dried, to various parts of the body. On the left side his responses were always given with confidence and correctly. On the right side they were hesitating and almost always incorrect and evidently the result of guesses. This impairment of temperature sense was most marked in the right leg, less in the arm, and not at all in the face.

The examination was frequently interrupted by the peculiar spells mentioned by the patient. The right arm would be abducted to the trunk, the forearm flexed upon the arm, and the wrist and hand flexed upon the forearm. The fingers, however, especially the index, middle and ring fingers, became rigidly extended. The movement was in no sense convulsive, but consisted of a slowly on-coming wave of tonic contraction, which gradually reached a maximum, maintained the same for the fraction of a minute, and

then gradually disappeared. It was distinctly athetotic in character.

The face presented no irregularity. The tongue was protruded in the median line. The ocular muscles seemed intact. On attempting roughly to study the fields, however, it was at once discovered that right lateral hemianopsia was present. On more detailed examination this proved to be sharply defined and typical. The pupils reacted well to diffuse daylight, but when the room was darkened and a candle used, they refused absolutely to respond as long as the flame was held upon the hemianopic side. As soon, however, as the median line was crossed and the rays fell upon the light-perceiving areas, they immediately contracted. The experiment was performed repeatedly and always with the same result.

An ophthalmoscopic examination failed to yield any evidence of choked disc. Both eyes, also, were excessively myopic.

Finally, jarring and percussion of the head failed to yield any evidences of pain. There was, in addition, no impairment of the mental faculties. He gave his history clearly, in well-chosen words, and answered all questions intelligently.

On February 26th I was again requested to see him. For six days past he had suffered severely from headache. The hemianopsia was unchanged, but there was now great loss of vision in the remaining half-fields. This he said had come about in the last five or six days. All of the other symptoms noted at the previous examination were confirmed. The weakness of the right side was now much more pronounced, and the spells of tonic contraction occurred every few minutes. The knee-jerk of the right side was found to be more exaggerated than ever, while the left remained about normal. The patient was very nervous and irritable, and evidently much worse.

On March 11th he was again seen. The weakness of the right side had increased and the impairment of sensation had become more marked. The knee-jerks remained about as before, but a decided ankle clonus had made its

appearance on the right side. Vision has become still more impaired on the sound side, but the Wernicke reaction was as marked as before.

Mental impairment was now quite evident. His remarks were frequently disconnected and at times silly and confused.

Taking the symptoms all in all, it is certain that a gross lesion of the left cerebrum was indicated, and further that this lesion was not cortical but involved the base, was evident from the presence of Wernicke's reaction. From the combined motor and sensory disturbance it was further evident that the lesion was more or less extensive, though the predominance of the sensory phenomena and the Wernicke reaction relegated it in the main, if not altogether, to the thalamus. Whether the pupillary reaction resulted from pressure upon the left of the tract or to disease of the pulvinar simply, was of course a matter of conjecture, though the first supposition, all things considered, seemed the more probable.

On February 28th, Dr. Charles A. Oliver made an exhaustive ophthalmic examination, which is herewith appended. The report is presented in detail, inasmuch as it presents a number of most interesting points.

From the statements of his attending physician, Dr. Gerhard, I learned that the patient gradually grew worse. His mental impairment gradually became more and more marked, and towards the last he was very somnolent. The weakness of the right side increased until loss of power in arm and leg was complete. This was true both for motion and sensation. There had been much general wasting, and in addition the temperature rose two days before his death to 104°. On March 24th it became impossible to rouse him, and on the following morning he died.

Autopsy.—March 26, 1890: Calvaria dense and heavy; dura decidedly adherent over vertex; veins of dura small; inner surface smooth and shining; longitudinal sinus not distended, contains a few small clots; escape of cerebrospinal fluid very small, some two drachms; pia-arachnoid somewhat opalescent and minutely injected; veins full, though not distended; membranes not adherent.

OPHTHALMIC EXAMINATION OF H. V. BY CHARLES A. OLIVER.

OBSERVATIONS.

<i>Right Eye.</i>	<i>Left Eye.</i>
1. Direct vision for form (corrected), reduced to V. = $\frac{5}{40}$.	1. Idem.
2. Power and range of accommodation, normal for age, and error of refraction.	2. Idem.
3. Right lateral hemianopsia.	3. Right lateral hemianopsia much more pronounced.
4. Remaining visual fields for both form and color, reduced to one-twelfth of normal areas.	4. Remaining visual fields for both form and color, reduced to one-seventy-second of normal areas.
5. Fleeting negative scotomata throughout the green color area.	5. More marked fleeting negative scotomata throughout the green color area.
6. Pupil four millimetres in horizontal meridian upon monocular exposure.	6. Idem.
7. Wernicke hemiopic pupillary reaction sign (which, more properly, should be called hemianopsic pupillary inaction sign) plainly manifest.	7. Hemianopsic pupillary inaction sign, but it is not so prompt to light stimulus as its fellow.
8. Extra ocular muscle action intact in all directions. (Slight insufficiency of the interni, to be accounted for by refractive error.)	8. Idem.
9. Ophthalmoscopically: the ordinary changes of high myopia.	9. Idem.
10. Retinal arteries and veins normal in comparative size, tortuosity, and tint of contained blood.	10. Both retinal arteries and veins enlarged, tortuous, and apparently carrying dark-colored blood.
11. A broad superficial splotch-like hemorrhage extending over the lower outer quadrant of the disc, and not seemingly connected with any retinal or choroidal stems.	11. No such vascular change.

RÉSUMÉ AND CONCLUSION.

The above series of differential symptoms may be summarized as follows:

1. Reduction of direct vision for form to one-eighth of normal on each side.
2. Right lateral hemianopsia more marked in the left eye.
3. Remaining visual fields of left eye six times less than those of the right eye.
4. Fleeting negative scotomata for green in green-color area more numerous and more pronounced before the left eye.
5. Hemianopsic pupillary inaction sign more marked in the left eye.
6. Retinal arteries and veins of the left eye enlarged, tortuous, and apparently carrying dark-colored blood.
7. A broad superficial splotch-like hemorrhage extending over the lower outer quadrant of the right optic disc, and not seemingly connected with any retinal or choroidal stems.

In this summary Nos. 2, 3, 4, 5, 6, and 7 point distinctly toward the left cerebrum as the situation and position of some coarse lesion, whilst No. 5 shows that the pressure is the greatest upon the left optic tract.

The ophthalmic symptoms in this case indicate a gross intracranial lesion involving the left cerebrum in such a position as to give the most pronounced intracranial pressure upon the left optic tractus.

Left parietal region of brain distinctly fuller than corresponding portion of right. This was noted both before and after removal from cranium. Brain quite firm to touch, preserves well its shape. Vessels of base normal. Small clot observed in basilar artery. Lateral ventricles not dilated, left in fact seems shallow. Walls normal in color, no injection of vessels. Choroid plexuses very cystic and dark. Velum interpositum dark red. Veins of Galen seem tense and full. Walls of third ventricle somewhat injected but otherwise normal. The thalamus and intra-ventricular nucleus of the left side were now carefully inspected. The thalamus, was much increased in size, though no marked change in its general configuration could be noted. It seemed simply distended, and in addition much more resistant to the touch. On section, a neoplasm, pinkish-yellow in color and quite firm, was revealed. It invaded chiefly the region of the pulvinar, though also to a considerable extent the tubercle. In the immediately adjacent portions of the caudate nucleus, it was also found, though to a much less extent. The internal capsule was not involved, unless perhaps a slight infiltration of the posterior third. Microscopically the tumor proved to be a glio-sarcoma.

Evidently the various symptoms presented by the patient were the result, first, of pressure of the growth upon surrounding structures; secondly, to a less extent the result of the irritation of these structures; and third, possibly to loss of function of the parts involved. To the first category belong, beyond a doubt, the various disorders of sensation, including the hemianopic pupil, together with the loss of power in the arm and leg. That marked increase of pressure had existed upon that side can be inferred from the post-mortem appearances, as well as from the eye-ground examinations. The peculiar athetotic attacks were probable due to irritation of the motor portion of the capsule. Lastly, to what extent the symptoms presented were influenced by loss of function of the parts involved must remain problematical.

It should be stated that microscopic examination of the quadrigeminal bodies, as well as of the optic tracts, was negative.

REPORT OF CASES ILLUSTRATING CEREBRAL LOCALIZATION.¹

By J. H. McBRIDE, M.D.,

Superintendent Milwaukee Sanitarium or Nervous Diseases.

THE following cases seem to me worthy of being placed on record :

CASE I.—*Sarcomatous tumor affecting the motor regions of both hemispheres symmetrically.*—Man aged twenty-eight, married, Bohemian. No history of syphilis or injury. The patient was first seen by me in January, 1888. His previous history was as follows :

Thirteen months previously his sight began to fail in left eye. In about two months sight began to fail in right eye. In six months was completely blind in both eyes. Within a few weeks of the time that his left eye began to fail, he first experienced pain in right knee and right foot. Shortly after this the pain involved the left knee and left foot, and for some time he was treated by a physician for rheumatism. Within one or two months after the pain began, he was not certain about the time, he said he had difficulty in walking. The right leg especially was weak, and he often stumbled and fell, although he walked with a cane. A few weeks after the right leg began to trouble him, he had difficulty in using the left leg. About three or four months later his hands began to grow weak, so that he could not handle his knife and fork or hold a cane. He could not remember that his hands and arms had ever pained him. His legs and arms continued to grow progressively weaker until I saw him thirteen months after the onset of the disease, when he was unable to stand alone. With a nurse assisting him on each side, he could walk across the room, but with a great deal of difficulty. While lying in bed he

¹ Presented to the American Neurological Association Annual Meeting in Philadelphia, June, 1890.

could use his hands for a great many purposes, such as picking up the covers of the bed and using a handkerchief, and while there was some inco-ordination, it was not very marked. He could carry an empty spoon to his mouth, but if it contained liquid always spilled it. In any attempt to use the hand there was a slight jerky tremor. In raising the legs from the bed this tremor was also quite marked, though much finer than that of the hand. He complained of frequent cramps in right arm and right leg. He stated that he had never had these cramps on the left side. Atrophy of the muscles of legs and arms marked. He could locate the position of his limbs correctly. Weights held in the hand were distinguished very imperfectly. Knee-jerk was exaggerated, ankle clonus always marked, superficial reflexes appeared to be normal. He said that during the previous two or three months he had had a constant feeling of pins sticking into his skin which affected the entire body and which persisted to the last. The dynamometer showed twenty for the right hand and nineteen for the left. During the previous three months he had convulsions one or twice a month. These convulsions seemed to be general, and he always lost consciousness; said he felt faint for about a minute before they came on, and always slept afterward. There was marked anæsthesia over the entire body; if anything it seemed to be worse in the legs below the knee. It was more marked in the right arm and right leg. He said that from the commencement of his disease he had severe headache every four or five days in the front and top of his head. This pain usually lasted about six hours and returned regularly every fourth or fifth day. There was partial paralysis of the muscles of the abdomen and spine, though it was difficult to decide upon the degree of their involvement.

At the time of my first visit he could not raise himself in bed, though he could sit up for a short time when once placed in a sitting position. The spinal and abdominal muscles evidently contracted feebly. Abdominal respiration was almost entirely abolished. From an early period of the disease he had to pass water frequently. It should also

have been stated that he had no aphasia and no paralysis of muscles supplied by cranial nerves. I did not see this patient during the last two or three months of his life, but obtained the facts of his later illness from his nurse. Three months before death he became completely paralyzed in his legs. Sphincters were also paralyzed. The paralysis of arms was progressive, and during the last few weeks of his life he could not raise his arms from the bed or move himself in any position. His mind, the vigor of which he stated was slightly impaired when I first saw him, progressively failed, the predominant condition being that of mental weakness. During the last few weeks of his life he was irritable and noisy. Six hours before death he had spasmodic twitching in left arm and hand which lasted a few minutes, then the right arm and hand twitched for about the same length of time. These twitchings of the arms alternated in this way for about an hour, at the end of which time he had severe general convulsions. After having had five or six of these convulsions within three hours, he died in one, November, 1888.

In regard to the convulsions it should be stated that with the exception of the twitching of the arms above mentioned, they always appeared to be general, though it was impossible to obtain from the nurse or from the patient any statement of the order of their development. I could not learn that they were at any time localized in their character, with the possible exception of the twitching of arms that occurred on the day of his death.

When I first saw him he was entirely blind, pupils were widely dilated and eyes staring. Looking at the patient from a distance, the eyes had the appearance of exophthalmic goitre. Examination by the ophthalmoscope showed that there was marked atrophy of both optic nerves. The patient said that he became blind in the left eye before the right.

On post-mortem examination the brain, including pons, cerebellum, and membranes, weighed forty-eight ounces. The skull was thickened in about the position of the anterior fontanelle. This thickened part was a little larger

than a silver dollar in extent. It was a little more than one-half inch in thickness in the centre, the bone being finely porous, or honeycombed. Just beneath this, and between the layers of the dura, was a deposit of sarcomatous material. The deposit was nearly circular in shape

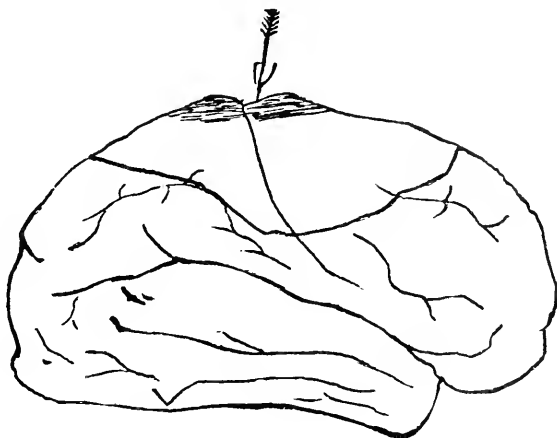


FIG. I.—Line shows approximate depth of tumor. Shaded portion shows superficial destruction of brain substance. Arrow indicates starting point.

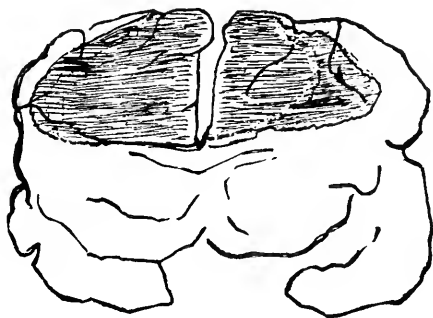


FIG. II.—Shaded portion shows depth of tumor in both hemispheres.

and was about one-half inch thick in the centre, shading off towards the edges. The dura in this vicinity was very much thickened, and was adherent along the margins of the hemispheres anteriorly and posteriorly. Neither the superior longitudinal sinus nor the falx cerebri were in-

volved. The upper end of the ascending convolutions in both hemispheres was involved in a destructive process, which superficially extended about two and one-half inches from before backward. In this region the brain substance in both hemispheres was destroyed, there being on each side an excavation into which the dura sank. On the right side this excavated place contained about an ounce and a half of recently coagulated blood. On perpendicular section of the brain the tumor was found to extend as low as to the upper border of the corpus callosum on the left side, and to within about one-half inch of the corpus callosum on the right side. It involved the convolutions on the left side as follows:

The posterior one-third of the first and second frontal, upper two-thirds of the ascending frontal and parietal, and upper one-half of the superior parietal lobule. On the right side the extent was almost identical, with the exception that not quite as much of the superior parietal lobule was involved. With the exception of the entire destruction of the upper one-third of the ascending convolutions the brain had the appearance exteriorly of health, and over all of the extent of the tumor, as here described, there was a mantle of apparently healthy tissue, the tumor being confined almost entirely to the white matter. On the right side, the median aspect of the ascending convolutions, the posterior one-third of the first frontal convolution, and a very small part of the precuneous were involved. On the left side, the extent of the tumor was essentially the same except that a little more of the precuneous was involved. The convolution of the corpus callosum was involved in about the middle one-half on each side, the diseased process extending, as stated above, to the level of the corpus callosum on the left side and to within one-half inch of it on the right. The middle region of both crura was softened, as were also both anterior pyramids. On examination with the naked eye, this descending degeneration appeared to be the same on both sides. Microscopic examination showed the tumor to be a small-celled sarcoma. The brain was accidentally thrown away, so that no further microscopical examination

was made, and the opportunity of studying the descending degeneration was lost, which in this case would have been a most interesting feature.

The above case seems to be unique in that the disease involved the two hemispheres almost precisely to the same extent and in the same region. An experiment deliberately conducted could not have been more accurately localized. Though my study of the case was obviously imperfect, there are certain points in localization upon which it bears and to which I will briefly refer. First, if we assume that the morbid process began in the upper part of the ascending convolution and worked downward (and this seems probable), then the first sensory and motor symptoms were referable to the involvement of this region, and the order of development of symptoms would correspond to the accepted views of the function of this region. Second, it will be observed that defective sight was the first symptom, though the disease was probably at that time superficial and in the upper part of the motor region. This simply emphasizes what has been noted by others, that impairment of sight may be the first symptom of cerebral tumor. Third, it should be noted that pain in the knees and in the feet was among the early symptoms pointing to a probable early involvement of the sensory centres. The patient was very positive about the existence of pain at an early stage of the disease. If the assumption is correct that the progress of the tumor was from above downwards, then it follows that the early sensory symptoms were due to involvement of the upper part of the so-called motor region, or rather of sensory centres in this vicinity. It should be remembered, however, that the convolution of the corpus callosum on both sides was involved. Fourth, according to Ferrier, irritation of the base of the first and second frontal convolutions in the monkey gives rise to lateral movements of the head and eyes, with dilatation of the pupils, and an expression of attention and surprise. In this case there was dilatation of the pupils and a staring expression that was always very noticeable, and it will be observed that the posterior part of these two convolutions was involved

in the morbid process. Fifth, the patient stated that his memory began to fail within about one year from the onset of the disease. During the last four months of his life there was, as stated, decided mental impairment. Was this due to a gradual involvement of the frontal region where some have located the higher psychical faculties?

CASE II.—Man aged seventy-three. He had been intemperate and was subject to chronic rheumatism and had an aortic murmur. On getting out of bed one morning he found that he was paralyzed on the right side, and fell to the floor. He could not speak until about noon, when, on seeing his son, he called him by name, saying, "This is 'P.' " During the day he tried to talk, but could only speak in an unintelligible jargon, except that once he said, "I will soon get well." He seemed to comprehend everything that was said to him, hearing and sight were normal, and he readily comprehended written and printed words. In the early part of the day the paralysis was incomplete, patient being able to move his arm and leg, though he could not grasp anything or stand alone. The paralysis grew worse during the day, and at five o'clock was complete. At time of examination in the afternoon there was evident anæsthesia of entire right side, though, owing to the mental condition of the patient its degree could not be determined. The face was drawn slightly to the left. He died on the same day the paralysis developed, at ten P. M.

On autopsy, the vessels at the base of the brain were atheromatous, having the appearance of a series of pipe stems. On looking at the left hemisphere, the operculum was lifted up, the convolutions of the island being plainly visible and bulging out. On section of the brain, a recent clot, weighing a little over two ounces, was found to be located chiefly in the convolutions of the island of Reil and outside of the claustrum. The hemorrhage was in the white matter and extended from about the middle of the second frontal convolution along the whole extent of the island, and extending to and involving a small part of the first temporal convolution. The white substance of the hinder part of the third left frontal convolution was also destroyed by the hemorrhage.

The interesting point in this case is the possibility of reaching the clot and evacuating it by surgical procedure. This could easily have been done if the lesion had been distinctly located during life. As part of the third left frontal was involved the aphasia could not have been cured by an operation, but as the paralysis and anæsthesia were due to pressure, upon the internal capsule, the evacuation of the clot would have relieved both.

THE PHYSIOLOGY OF SLEEP.

An editorial in "The Medical Age," March 25, 1890, contains the following: The sleep state follows that fatigue, which is the expression of waste, unbalanced by repair. There is a diminution of cerebral molecular energy, owing to diminution of force-giving intra-cellular material. All the brain functions are languidly performed; the heart and the lungs participate in the lessened production of force; the blood contains an excess of carbonic acid, which increases the depression by hindering metabolism. We are ignorant of the mechanism and only know that it comes about through fatigue. According to Preyer it is an intoxication from retention of waste products, one being lactic acid. This acid is not the only poison produced in the system, for every cell is a laboratory yielding *leucomaines*. These waste products are found largely in the urine, and when this secretion was injected into animals, Bouchard found that the urine of the day was twice as toxic as that of the sleeping state; the former producing narcosis, and the latter convulsions. The inference is that during waking activity, disassimilation yields products which by accumulation cause sleep, and during sleep the convulsivant substances resulting from denutrition bring about awakening. Pflüger also connects sleep with the process of disassimilation. His theory is that brain work requires nutrition and oxygen. During waking hours, waste gets ahead of repair, and carbonic acid and other products accumulate in the system. The oxygen in the cerebral cells combining with the carbon of albuminoid matters causes little explosions from which arises the waking state. When the oxygen of the tissues diminishes by reason of its consumption, the cause of the excitation is lacking and sleep occurs.

A CONTRIBUTION TO THE THERAPEUTICS OF SPASTIC PARALYSIS.¹

BY V. P. GIBNEY, M.D., NEW YORK.

THE etiology and pathology of this affection have been so fully elaborated that I shall make no attempt to discuss either one of these points, but will present for your consideration a few cases by way of illustration. The treatment I have adopted is what is known as the "Orthopedic Treatment," and, as the neurologist generally refers the management of these cases to the orthopedic surgeon, it may be of interest to the gentlemen present to know just what can be accomplished.

CASE I.—A girl seven years of age, referred to my clinic by Dr. Hogan of New York, in April, 1887. She was very irritable, cried on the slightest provocation, was totally unable to walk or to stand alone, but could with assistance manage to get across the floor in a scissors-like kind of gait. She would balance herself on her toes and balls of the feet, and make an attempt to walk by bringing the feet forward, one foot overlapping the other, the thighs, of course, being in marked adduction and knees in flexion. She had begun even this kind of walk only a year or two previously. She was the only child living out of six. Two of this number were still-born. This child was born at full term, and was thought to be perfect. When five months of age she had convulsions lasting three or four days. When the convulsions subsided it was thought that she was totally blind. She lay in a kind of stupor for nearly three months, and had a slight convulsion at the end of this time. Six months later her sight began to return.

Under chloroform, I divided the Achilles tendons subcutaneously, the adductors of the thighs and the ham string tendons on each side. The adduction was further overcome by force, knees straightened, and the feet placed in position of slight calcaneus. I placed the child in a wire

¹ Presented to the American Neurological Association Annual Meeting in Philadelphia, June, 1890.

cuirass, the leg portions of which were abducted. She was taken to her home in Harlem, and several doses of opium were required to make her comfortable. She was kept pretty snugly encased in the wire cuirass for six weeks. On removing her from the apparatus, the ham strings were found not fully stretched. Posterior braces were applied, extending from the thighs to the calf, and with these I succeeded after a month or two in overcoming much of the contraction, nearly all. At the time of the operation I ordered potassium iodide in increasing doses taken in milk or vichy. She reached forty grains three times a day within a month. During the summer I had the mother employ massage as best she could, and forcible stretching of the adductors and ham strings. The feet were in very good position.

In the fall of 1887 she was walking about unassisted. Her excitability had diminished about seventy-five per cent. When last seen, about six months ago, she was able to walk several blocks, got her heels well down to the ground, and the in-knee was very slight.

CASE II.—My friend, Dr. Bunker, of Brooklyn, referred a boy eight and a half years of age to me on December 11, 1885. He suffered then from an acute synovitis of the left knee, but I found on examination that this was a typical case of spastic paralysis, or contraction. The synovitis was treated by rest and compression to the parts, and in a month he was cured of this. I saw him some months later. No relapse had occurred, but his limbs were much distorted at hips, knees and ankles. I suggested a course of treatment for him, but it was not until the 14th of October, 1889, that he was placed under my care for treatment of his spastic condition.

Both feet were in marked equino-varus, so that he stood on the outer borders of the feet. The adductors of the thigh were in moderate spasm, but would yield quite readily to a little force. The leg flexors were tense and quite resistant. By employing all the force he would submit to, I could extend the legs to 175° . Beyond this point, however, marked reflex spasm was excited. The Achilles tendons on both sides were tense.

On the 14th of December, under ether, the tense bands in popliteal space were divided subcutaneously on both sides; the knees were fully extended, but not hyper-

extended; the tendo-Achillis on right side was divided, and foot forced into position of hyper-flexion; the tendo-Achillis on left side was also divided, as well as several bands of plantar fascia; the foot was placed in position of calcaneo-valgus. I found it unnecessary to divide the adductors. He was put up in plaster of Paris from the hips down to the toes. On December the 8th a note was made that he had suffered very little since the operation, complaining only occasionally of pain in the knees. After two or three weeks in this position, I was able to extend the legs still further, and, after retaining them in an over-corrected position for a few weeks longer, I had a masseur take charge of the case under my direction. This treatment was continued for about a fortnight three times a week. On the 21st of April, 1890, I made a note that he could flex the foot up to the normal extent voluntarily; that he was walking a few steps alone; could voluntarily extend his legs to 175° ; they could be passively hyper-extended. I had him wear club-foot shoes to keep his feet in good position by night, and had the ordinary leather shoes built up on the outer side for use by day.

On the 29th of May, present year, I received the following note from Dr. Bunker: "You should know that Charlie Miller (the patient whose case I am now reporting) goes without crutch or stick, walks out with the girls, and plays lawn tennis."

CASE III.—On the 20th of June, 1889, my friend, Dr. Gray, of New York, referred to me a case of talipes equinus (spastic), in a girl five years of age, living in Brooklyn. She had begun to walk about a year previously, but had gained very slowly. She walked like a prancing horse, was very excitable, and had very little control of her emotions. There was no adduction of the thigh, very little contraction of the leg flexors; speech very imperfect. The Achilles tendons were short. They were both divided, and feet put up in position of over-correction. She progressed without any special incident worthy of note, and on the 2d of August she was able to stand with heels squarely on the floor, and could walk without braces or shoes very much better than she could at first. She wore club-foot shoes for several months, and, in February of the present year, the club-foot shoes were removed, and she was provided with a

stout pair of leather shoes. On the 29th of May, present year, I made a note that she was walking very well indeed, and that her inco-ordination was comparatively slight.

CASE IV.—A boy eight years of age was admitted to the Hospital for the Ruptured and Crippled, October 10, 1889. Diagnosis: spastic paralysis. The history given was this: That before he was a year old the mother noticed that his limbs were inclined to cross and that he was unable to sit alone. When four years of age he began to walk a little, but the limbs were so badly distorted and his inco-ordination so great, that he required assistance. Even then he did not get his heels to the floor. The right side was more distorted than the left.

A few days after admission I put him under ether, divided the adductors, ham strings and Achilles tendons, and put him up in plaster of Paris, with thighs well abducted, legs extended and feet flexed beyond 90°. The case progressed slowly, plaster was not removed for four or five weeks, and then an excoriation was found over the sacrum. It was necessary after removing the plaster to apply knee braces and to support the feet with apparatus without any joint. On the 30th of January he was discharged from the hospital, barely able to stand alone and unable to walk. He went into the country, and his mother was instructed very particularly about the management of his limbs. He did unusually well. I saw him about a month ago, and was surprised to find how straight the limbs were and how he had improved so far as his inco-ordination went. The Achilles tendons were of normal length, muscles were developing, and, while I am unable to give his condition at the present time, I feel confident that he will be able to walk and get about with comfort.

The cases just reported are a small number of those I have treated in this way for several years past, and, while many fail to get the benefit that these here recorded have received, I am confident that many have been enabled to walk and get about without assistance. It is necessary after dividing the tendons to keep the feet in normal position for two or three months after the operation. The tendons are apt to lengthen, and calcaneus has in some instances resulted. The relief given to the nervous condition in many of these patients is most marked.

A CASE OF INSULAR SCLEROSIS IN WHICH AN
ATTACK OF CEREBRAL HEMORRHAGE
ARRESTED THE TREMOR ON THE
HEMIPLEGIC SIDE.¹

BY WHARTON SINKLER, M.D., OF PHILADELPHIA.

SAMUEL SMITH, laborer, aged sixty-four, Ireland; one of five children; father died of phthisis at fifty; all of his paternal ancestors, it is stated, died with this disease, including two brothers and four sisters. His mother died at an advanced age; cause unknown. One sister died of some disease peculiar to women.

He is a man who has always enjoyed good health. Some twenty years ago he experienced some difficulty in micturition, but all venereal disease is denied. He has drunk beer freely, but not whiskey. He had small-pox at four years of age; typhus fever at ten.

At about ten years of age he was working in a silver-plating shop, where he inhaled the fumes of ammonia and muriatic acid. He had been working there for fifteen years when he began to notice, on holding a glass, a tremor in both hands. This tremor began simultaneously in both hands, but it was more marked in the left. This ceased entirely when the hand was at rest. The tremor incapacitated him from occupations where fine movement was required; but he experienced no pain, and regarded himself in perfect health.

Three years ago he was in the Philadelphia Hospital for this tremor. He remained five months, and considered himself much improved. Diagnosis at that time: *insular sclerosis*.

September 14, 1889, while working on a farm, after exposure, he suddenly, one afternoon, without loss of con-

¹ Presented to the American Neurological Association in Philadelphia, June, 1890.

sciousness, became totally paralyzed on the left side; speech unaffected. In six weeks power began to return in arm, and in the course of three months in leg. Since the seizure on September 14th there has never been any tremor on the left side.

At no time has the head been involved. Tongue on protrusion is drawn to the left; it exhibits a fine tremor. There is at present a coarse tremor of the right hand, which disappears when at rest, and is much exaggerated when the fingers are separated and when any movement is attempted. The right leg is uninvolved. No tremor can be detected in the left arm. The patient can carry a tumbler of water to the mouth without spilling any of the contents. This feat cannot be accomplished with the right hand. There is no nystagmus, apparently, in the eyes; pupils are unequal, the left being the larger: they respond to light and distance. The left leg is raised with difficulty. On the right side the patellar reflex is apparently normal; on the left side it is much exaggerated. There is also a marked muscular irritability to mechanical strimulus. No ankle clonus can be developed on either side.

The power in the hands is fair, the dynamometer giving 80 in the right hand and 40 in the left. There is a clonus present in the left forearm.

Sensation is well preserved throughout the body. There is constipation continually. Micturition is normal. The sounds of the heart are free from murmurs, but seem poor, especially the second, which at the base is somewhat accentuated.

It should be stated that, while working in the silver-plating works, he lost some of his teeth.

He was subject, at infrequent intervals, to slight attacks of vertigo previous to his hemiplegic attack. These have all disappeared.

At present his appetite and general health are excellent.

THE HYPNOTIC STATE OF HYSTERIA.¹

BY WILLIAM C. KRAUSS, M.D., BUFFALO, N. Y.

THE seeming mysticism which has enveloped a certain case, some thirty miles east of Buffalo, has attracted widespread attention, not only in the professional, but in the secular press as well. Hints at witchcraft, sorcery, etc., have been enounced by those who have watched the progress of the case, as recorded in the local papers, and this feeling has taken some root among the incredulous and skeptical. It is to clear up and efface some of this feeling that I present this paper to the Association, and hope I may be able to throw some light upon a case, not unparalleled, as many suppose, but well known and definitely placed in our nosology.

The first reports of the case reached me while attending the nervous clinics of Prof. Charcot at the Salpêtrière, Paris. I had not long to wait before seeing cases similar to the one in question, and noted carefully all symptoms and modes of treatment, hoping that on my return I might be able to apply them. Although acquainted with the family for years, my endeavors to gain the case proved unavailing. The report of the case, therefore, is based upon material furnished me by Editor Landsittel, of the "Attica News" (who was a trusted and confidential friend of the family), and by observations which I made during my several visits to the patient:

Emma T. Alt: age, twenty-six years; married March 6, 1886; two children, one living and one dead; height, five feet three inches; weight, 160 pounds, before taken sick; complexion, fair; hair, dark-brown; constitution, strong, vigorous, healthy.

Antecedents.—Parents came to this country from Northern Germany thirty-five years ago. Paternal side free from

¹ Presented to the American Neurological Association at the Annual Meeting in Philadelphia, June, 1890.

any hereditary taint; maternal side has neuropathy plainly stamped upon it.

Early History.—Patient passed through the common infantile affections without any apparent sequelæ. She received a limited education, and was considered very apt in her studies; was employed for some years as a domestic, and was regarded as a quiet, unassuming young lady. Previous to her taking sick, she passed through much mental and moral excitement, which doubtless laid the foundation for her future condition.

About the middle of August, 1887, she was taken ill, as the physician then reported, with a "severe dysentery, followed by inflammation of the bowels and stomach and the formation of abscesses." When these abscesses were relieved she usually fell into a sound sleep, lasting about three days. She would then lie partially awake for a day and a night, and then gradually sink back into another sleep of from five to seven days. The length of these sleeps increased to ten to twelve and more days, and on February 27, 1888, she awoke from a seventeen days' sleep. It was during this last sleep that her case attracted attention, and was first reported in the local paper ("Attica News," February 24 and March 2, 1888). During the trance she is described as "lying on her left side, with her right arm slightly thrown up over her head; she lies perfectly still, save a slight delirious waving of the upturned hand and at times a tremulous movement of the upper eyelids. Her lips are continually moving in soft whisperings, uttering passages from sacred songs, etc. There are hours of inanition, when the hard breathing, the movement of the hands and eyelids, and the mutterings cease altogether." At the hour predicted she awoke from her slumber, and appeared to be in good humor for some time. When questioned as to what transpired during her sleep, she could not reply.

On February 28, 1888, she fell into another trance, as predicted by her. "Her right hand, warm and active, is thrown over her head, while her left hand lay upon the counterpane, white, cold, and lifeless. Her pulse is scarcely perceptible, respirations heavy and regular, and the motion of her right hand is strong and regular as clockwork." She awoke after three days.

On March 11th she fell into her predicted thirty days' trance. "She is now perfectly still, her body and extremities are cold and white, while her face is flushed. Pulse, 65 per minute and very feeble." During this period visitors were admitted into her room and tried to awaken her by pinching and pricking the skin, and as a consequence her

arms and neck are covered with black and blue spots. All attempts to awaken her from this sleep were unavailing.

On April 13, 1888, at 8.25 P. M., she emerged from her long sleep. Just before awakening she was taken with epistaxis, rise of temperature, and a strong pulse, which continued for a few hours, then disappeared. During the interval of rest she neither closes her eyes by night nor day, has no appetite, and her attitude becomes rigid, save the movement of the eyelids.

After a few days she fell into another sleep of short duration, and on awakening was taken "with a severe coarse cough, could not speak above a whisper, and respired with much difficulty."

April 7th she fell into another sleep of three days' duration.

April 12th she had another trance of two days' duration. From this time on, without any apparent cause, her attacks of *sommeil* (as the French term it) have been wearing off, her appetite is improving somewhat, and she shows signs of general improvement.

Some of the minor points I have purposely omitted, as they have no bearing upon the truly scientific part of the case and would only serve to confuse the clinical history. The story of her being poisoned, and the appearance of hemorrhagic spots upon her body (which make their appearance but once a year, generally on June 15th) as it occurred in 1888 and 1889, has also been ignored.

My first visit to the patient was made in September, 1889. I found her thin, pale, and haggard, quietly reposing upon her bed, surrounded by much medicine and many attendants. The room was closely curtained, with a temperature nearly that of body-heat, and her immediate vicinity showed that trim and tidyness so characteristic of such mental states. Her mind was unobscured, her memory seemed unimpaired, and she appeared to have full possession of her mental faculties. Her face and hands showed no deformity, her tongue did not deviate from the median line, and the orbits were normal in their movements. The grasp of her hand was feeble. I could not examine the lower extremities, but was informed that she had no motion in them; whether this was due to inanition or hysterical paraplegia, I cannot say.

Her voice was very feeble and not above a whisper, showing marked aphonia. Her respirations were frequent and superficial, dyspnœic in character, analogous to tachypnœe. Pulse small and feeble, 85 beats per minute. Temperature did not seem to be increased. Ageusie, anosmie, and ocular troubles had existed, but were now much improved. A careful examination of the general sensibility could not be made, but the arms and face showed no anæsthesia. Ovarialgia was wanting, as were other hysterogenic points.

A searching inquiry into her condition during the trances elicited the following: She generally fell asleep and awoke at nearly the same time, viz., 8 o'clock P. M. On some occasions she would awaken after an epistaxis or slight convulsion. While asleep her breathing was inaudible, not stertorous. Her food (which consisted of milk) was administered to her by forcing open her teeth and allowing it to run into the mouth from a spoon. The amount was variable: some days but a few teaspoonfuls were retained, at other times more.

During the periods of slumber she neither urinated nor defecated, but just before awakening incontinence would set in. At times, during these trances, she would have fainting-spells, convulsions, and, on one occasion, fell from the bed, inflicting some injury to her right arm.

Recently admission to see the patient has been denied me, and I cannot report her present condition.

On June 15th, 1890, I had occasion to see the patient, having previously been informed that the 'black and blue spots' had reappeared that morning. I found her apparently in the same general condition as at my last visit, some months ago, but on interrogating elicited the following history: At times during the day she is able to sit at a window for a few moments, and appears to be gaining in strength. Her food is limited to milk, of which she takes a small quantity daily. Instead of being in a condition of hypnosis, she is now prone to prolonged wakefulness, sleeping from two to four hours in every forty-eight hours. That morning (June 15) about 1 o'clock, she complained of

a burning sensation about her arms and body, followed by swelling, so that her sleeves had to be opened. About 3 P. M. that day I found her arms and body covered with large plaques, purplish blue in color, somewhat raised, but without pain or pitting. The extravasated portions were somewhat warmer than the surrounding parts, did not disappear on pressure, not symmetrical, but diffused over the arms and neck and chest. This condition lasted 2 to 3 days, and then disappeared—no doubt ready to reappear June 15, 1891.

The clinical history of the case resembles strikingly two cases which I had the pleasure of observing at the Salpêtrière, Paris. These cases, familiarly known as "dormeuses," were only conditions or states of that extensive nervous affection, hysteria, and were designated by Charcot as the "hypnotic state of hysteria." Charcot contends that this state is nothing more than "hysterical convulsions," "transformed" or "modified," or the "equivalent of a series of convulsive hysterical attacks." In such cases the attacks occur so frequently that the patient, before leaving one, is seized with another, and these continue almost *ad infinitum*. Attacks may follow each other with surprising alacrity. In a case studied carefully at the Salpêtrière, a series of 8,000 attacks occurred in fifteen days, and 21,708 in twenty-six days. In another case 4,506 attacks occurred in thirteen days; another had 17,083 attacks in fourteen days. In these cases, the attacks becoming modified by frequency, make up one long attack, lasting from days to weeks, and finally terminate or are transformed into hypnosis. Whether this theory be the correct one or not, it is very evident that these hypnotic states differ materially from induced hypnosis, on the one hand, and normal sleep, on the other.

Characteristic of the hypnotic state of hysteria are: 1, history of hysteria; 2, trances of variable duration; 3, complete resolution of all tissues of the body; 4, inability to rouse except in those patients offering ovarialgia; 5, absence of stertorous breathing; 6, marked diminution or suppression of the excreta; 7, oscillatory movements of some portions of the body; 8, capacity to forego alimentation; 9, masked convulsions; 10, periodicity.

1. *History of Hysteria*.—Charcot states that they are nearly all subject to grand attacks, and in some cases these attacks of hystero-epilepsy are transformed into attacks of hypnosis. The case in question did not develop hysteria until after she became the victim of circumstance. It then progressed rapidly. I am informed that she was never subject to grand attacks, the symptoms choosing some other way to disclose themselves.

2. *Trances of Variable Duration*.—These may last from a few days to weeks, months, and even years. The longest trance in the above case lasted thirty-two days. One case that I observed at the Salpêtrière was asleep forty days; another, twenty-nine days. Charcot has observed some lasting very much longer.

3. *Complete Resolution of all Tissues of the Body*.—The sensory, motor, nutritive, and other systems of the body seem to be in a state of stupor and their normal physiological action reduced to a minimum. This condition has been likened to the hibernation of some of the smaller mammals, characterized by a long period of stupor and torpidity, lasting through the winter months. Exceptions may occur in some cases, where contraction of some member may persist throughout the hypnosis. Otherwise the members are limp and anæsthetic, but retain their normal temperature and appearance.

4. *Inability to rouse the Patient except when offering Ovarialgia*.—All methods calculated to rouse one from a deep normal sleep prove unsuccessful in these cases. Faradization, inhalation of strong ammonia, pinching and puncturing the skin and nerves, do not cause the slightest modification of the physiognomy. In those cases offering ovarialgia or other hysterogenic points, the key to the situation is generally present. It is well known that digital pressure over these points may precipitate an hysterical attack, and also abort one. In like manner this procedure may throw the patient into a trance, and, on the other hand, may rouse him from his slumber. In some cases only a partial awakening is obtained, the patient uttering some cry and then relapses into his former condition.

5. *Absence of Stertorous Breathing*.—This, so common in normal sleep, owing to relaxation of the velum palati, is always absent in hysterical slumber, and serves as an aid in distinguishing the normal from the abnormal sleep.

6. *Marked Diminution or Suppression of the Excreta*.—Since the organs of the body are in a state of stupor, it follows that their functions are also impaired. The processes of nutrition, assimilation, oxidation, etc., are very sluggish and inactive, and, as a result, the excreta are notably diminished. In the case in question it is stated that while asleep no urine or fæces passed from her, either voluntarily or involuntarily. Generally the urine is very much diminished, both qualitatively and quantitatively. A short time before awakening, the urine increases in quantity and the salts reappear in their full strength. This is generally taken as an index of a speedy awakening, and can in most cases be relied upon.

7. *Oscillatory Movement of Some Parts of the Body*.—Generally noticed in the upper eyelids, and on attempting to open the lids the vibrations become more pronounced. The hands may also become tremulous—as in the above case—resembling very much the tremor of paralysis agitans.

8. *Capacity to forego Alimentation*.—The comatose condition in which these patients seem to be really calls for little nutriment to supply the waste of tissue which is slowly going on. Loss of weight is generally noticed in every case, but is not in direct ratio to the amount of food ingested and the duration of the trance.

9. *Masked Convulsions*.—In one of Charcot's cases the patient while asleep would raise the body to the sitting posture, then fall back into her former position, at the same time muttering some inaudible sentences. In the case in question, "fainting-spells" occurred quite frequently while asleep, followed by convulsions and mutterings, which were of a religious nature. The pulse and respirations during the trances remained normal—the same as occurs during the grand attacks of hystero-epilepsy.

10. *Periodicity*.—In hysteria, periodicity plays an important role, and may almost be called pathognomonic of this state. The time for passing into or out of the trances in every case occurred always at or near 8 o'clock P. M.

The prognosis and treatment of these cases is that of general hysteria.

CEREBRAL GUMMA SIMULATING THE SYNDROME OF GENERAL PARESIS.

The "Annales Médico-Psychologiques" for March, 1890, contains clinical archives of particular interest, notably the history of the condition mentioned above. A cerebral tumor may give rise to symptoms that belong to general paresis, as loss of intellect, difficulty of speech, inequality of the pupils, and thus confuse the diagnostician. Syphilitic lesions of the brain, vascular alterations of its coverings or substance, cause diffuse periencephalitis and bring about clinical symptoms of general paresis. Zambaco cites a case where manifestations of general paresis appeared twenty years after a chancre and syphilides. The autopsy revealed a gumma the size of a hazel-nut in the anterior portion of the frontal lobe, near the median line. The entire brain was softened, particularly in its anterior lobes. The ventricles contain an enormous amount of serous fluid. But there are subtle differences between real and simulated general paresis. The syphilitic is idiotic, brutalized, and prematurely demented; his ideas of grandeur and ambition are less expansive; his egoism is less; he is more reserved and timid. The tremor, which consists in a trembling and a fibrillary, vermicular movement of the lips and tongue—almost pathognomonic of general paresis—is comparatively slight and sometimes absent among syphilitics. Tremor in the hands is frequent, though not of the same degree as in general paresis. Motor disturbances, partial or general, exist earlier in syphilitics than in paretics (Manriac). The following is the summary of a case in which certain neurologists—Magnan, Maradon de Montyel—gave a diagnosis of general paresis associated with hypochondria:

Louis A., twenty-nine years, cook; impairment of intellect; ideas of ambition and hypochondria; difficulty of speech; inequality of the pupils; excitement, especially at night; food refused; death from marasmus. Autopsy: meninges not adherent, but gumma in the left hemisphere.

Periscope.

DRS. LOUISE FISKE-BRYSON, A. FREEMAN and GRACE PECKHAM.

ATAXIA IN A CHILD OF TWELVE.

The "Montreal Medical Journal," March, 1890, contains Dr. Blackader's report of such a case, W. J., a boy, the second child in a family of ten. Seven of these children died of infectious disease, with the possible exception of one infant who succumbed to diarrhœa. The other two survivors are a girl about four and an infant of sixteen months, who are in good health and without impairment of knee-jerk. The family history, near and remote, is good, except an occasional excessive indulgence in alcohol on the part of the father. W. J. was quiet as a baby; nursed till the tenth month; a little late in teething; no convulsions; but had a fall, cutting the forehead, the scar being still perceptible a little to the right of the median line. Since the age of five he has suffered from headaches on the right side, resembling migraine, accompanied by vomiting, which lasted several hours and pass off during a night's sleep. Any excitement may produce them, though their regular appearance is three or four times a month. They are less severe than formerly. The first unsteadiness in gait was noticed about six years ago, and the alteration in speech began two or three years ago, gradually becoming more marked. Present condition: fairly well-nourished, four feet five inches in height, weighs eighty-five pounds, has distinct talipes equinus in both feet, slight curvature of the spine, absence of patellar reflex, slight diminution of cutaneous reflexes, well-marked ataxic gait, and ataxia of the upper extremities. The body sways from side to side in walking, the legs are widely separated, and the feet thrown forward. Even when standing, there is much swaying if the feet are placed together; and this swaying is only slightly increased by closing the eyes. Walking backward is fairly well done. Attempts to touch the tip of the nose are about an inch or two at fault, though picking up a pin, even with the eyes closed, meets with greater success. There is no muscular atrophy, and no spastic rigidity. Speech is jerky, with an abrupt pause between the several syllables, and occasionally elision of the last

consonant, all of which is less marked in reading aloud than when speaking. There are no abdominal or thoracic symptoms; urine normal; no constipation; sleep generally quiet; no nocturnal enuresis. His mother states that it takes him longer to void urine than formerly. Pulse standing), 84—regular. Intelligence unimpaired. Ophthalmoscopic examination: "Vision normal; accommodation active; pupils even, oscillating three mm.; color vision normal; field free; fundus, slight posterior staphyloma, vessels slightly smaller than usual; no nystagmus, but some slight ataxia of muscles of eyeball."

During the past few years Friedreich's ataxia has been fully recognized by the profession, cases being recorded from time to time in the journals, so that the salient points of difference between it and Duchenne's tabes dorsalis are now generally acknowledged. Onmerod ("Brain," vol. vii., p. 111), Carré, Dreschfield, and Powers have reported cases. The symptoms in his patient, Dr. Blackader thinks, point to disease confined almost entirely to the posterior columns, but involving also the medulla. Cerebellar trouble would seem to be excluded by the history, by the absence of occipital pain and of optic neuritis, the absence of patellar reflex, and the presence of ataxia in the upper extremities. True tabes is also excluded by the age (six years) when the disease appeared, the impairment of speech, the absence of lightning pains and of alteration in pupillary reflexes. Insular sclerosis would give somewhat similar symptoms. The absence of eye symptoms and any paresis or spastic rigidity would exclude such a diagnosis. From other cases of Friedreich's disease, this differs in the following particulars: the absence of any other known case in the family, the previous migraine, and the indications of some paresis of the bladder.

SYPHILIS AND GENERAL PARESIS.

The "Bulletin de la Société de Médecine Mentale" for March, 1890, contains Dr. Cuyllit's paper on this subject. Three hypotheses exist concerning the relation of syphilis and general paresis. One, that general paresis always has syphilis as a cause. Another, that syphilis never causes it. And the third, that syphilis exercises some influence upon its production, whether as determining or occasional cause. There is still another view to take of the case: general paresis engenders syphilis. The general paralytic during the stage of excitement that precedes the development of his true malady gives way to every kind of excess, especi-

ally sexual excess, and is particularly liable to contract syphilis. Thus, from being paralytic, he becomes syphilitic. An interesting case is cited where general paresis of the insane followed the tertiary stage of syphilis, ten years after the first symptoms of specific poisoning. This late development is the rule. A non-syphilitic sister of the patient was also insane, suffering from ideas of persecution. While paresis may follow syphilis in eight or ten years, it does so only when one powerful aid is present—unsoundness of the nervous system, or heredity. As Dr. Régis puts it: "To manufacture general paresis, inoculate the sons of apoplectics with syphilis." Alcohol, tobacco, and sexual excess may bring about paresis in the nervously unsound. Is not persistent dissipation an evidence of a depraved nervous system? The record of thirty cases of general paresis appearing at the asylum of Everre during a few months, may be arranged as follows:

Men.	Women.	Doubtful or non-syphilitic.	Syphilis probably.	Syphilis.
19	11	5	5	9
11		4	3	4

THE ETIOLOGY OF EPIDERMIC HERPES ZOSTER.

The "Gazette des Hôpitaux" (Feb. 25, 1890) contains a review of Gautier's ideas upon this subject. The author thinks it a general infectious disease, of limited contagiousness, falling in particular upon the intervertebral ganglia, and producing its eruption, as other erythematous general infectious diseases, certain poisons, such as carbon oxide and arsenic also produce herpes zoster.

Predisposition plays an important role in its production. Three-quarters of the number attacked are the victims of arthritism—the disease of retarded metamorphosis.

A few things yet remain to be done: To isolate the pathogenic micro-organism; to make cultures of it; to inoculate the sound and produce herpes zoster by this means.

CULLERRE'S CLASSIFICATION OF INSANITY.

In "Brain," January, 1890, there is a careful review by E. Birt, of Cullerre's "Traité Pratique des Maladies Mentales." The author of this manual is medical director of the lunatic asylum at Roche-sur-Yon, and his treatise is an exposition of psychological medicine as taught by the present French school. The system of classification of insanity that he adopts is based upon the French classic model, comprising three great divisions, viz.: (1) primary alienations; (2) alienations associated with organic cerebral

affections; and (3) alienations associated with various morbid conditions. The divisions and subdivisions stand thus:

I. Primary Alienations.	1. Simple In- sanity.	{ Mania. Melancholia. Periodic Insan- ity. Progressive systematized delirium.	{ Intermittent Insanity. Insanity of double form. Circular Insanity.
	2. Degenerative Insanity.	{ Hereditary In- sanity.	{ Mental <i>obsessions</i> ; irresis- tible impulses. Systematized delirium of hereditary cases. Reasoning mania, moral insanity. Polymorphic deliria.
	3. Idiocy.		
II. Alienations associated with organic cere- bral affections.	{	{ General paralysis (diffuse interstitial encephalitis. Senile dementia (cerebral atheroma with consecutive atrophy). Organic dementia (focal lesions).	
III. Alienations associated with various gener- al morbid conditions.	{	1. With neuroses.	{ Epilepsy. Hysteria. Chorea.
		2. With poisons.	{ Alcoholism. Saturnism. Morphinism. Pellagra. Cretinism.

The form *dementia* is discarded on the ground of its being only a secondary condition, a mode of termination of insanity, properly speaking, and no longer recognized as a special form of mental disease. The group *monomaniæ* is replaced by *hereditary insanity*, which substitutes for simple disconnected psychological entities the notion of a permanent affection of the nerve-centres, having diverse manifestations according to individual circumstances, but connected one to the other by the continuous bond of degeneracy. The above nomenclature, says the reviewer, does not differ materially from that recently submitted to the Medico-Psychological Association of Paris for adoption as the groundwork of an international classification. In certain particulars it contains evidence of a decided advance upon the systems found in the generality of English textbooks.

SOME CEREBRAL LESIONS.

The first of the Goulstonian Lectures, with the above title, by Dr. G. Newton Pitt, appeared in the "British Medical Journal," March 22, 1890. It is an analysis of fifty-seven fatal cases of ear-disease, and of the complications that led to death.

CANNABIN IN EXOPHTHALMIC GOITRE.

The "Canada Medical Record," March, 1890, prints the following formulæ, recommended by Valieri for exophthalmic goitre ("Weiner Med. Presse," No. 41): 1. Four grains and a half of canabin with sugar of milk to make five pills; the pills to be taken in twenty-four hours. 2. Four grains and a half of cannabin, one ounce of syrup of orange, and three ounces of distilled water, mix together; to be taken in teaspoonful doses in twenty-four hours.

CLINICAL REMARKS ON PSEUDO-PERITONITIS AND
EPILEPSY IN HYSTERIA.

The "British Medical Journal," February 22, 1890, contains Dr. John Syer Bristowe's paper with this title, which is devoted to the narrative of two cases.

CLINICAL PSYCHIATRY.

"Montreal Medical Journal," March, 1890, calls attention to the fact that active steps are being taken to establish, in London, a hospital for the treatment and teaching of all that pertains to insanity. In connection with it, there will be a staff of thoroughly experienced physicians, whose duty it will be to promote the scientific study of mental disease. This movement will also enable future practitioners to see and know something of mental disorders before graduating.

HYPODERMIC TREATMENT OF ASTHMA WITH STRYCHNINE
AND ATROPINE.

In the "Boston Medical and Surgical Journal," April 3, 1890, there are records of asthmatic cases treated with atropine and strychnine hypodermically, by Dr. Thomas J. Mays. To alleviate an attack of asthma and prevent its recurrence, various drugs, such as morphine, lobelia, stramonium, chloral, chloroform, nitroglycerine and pilocarpine have been used, together with the employment of measures that tend to break up the abnormal causal connection that exists between other organs and asthma. The disease is essentially a spasmodic neurosis of the pneumogastric. Disorders of all the organs supplied by branches of the pneumogastric nerves are most liable to excite an attack, though without the peculiar predisposition, it is doubtful if such results would follow. A general lowering of nerve-tone is as much a cause as diseased organs. Invigorating the nervous system may banish

asthma as well as other abnormities. The possibility of strychnine hypodermically was first suggested by the excellent results Dr. Echeverria obtained through its use in epilepsy. Theoretically, remedies for epilepsy should relieve asthma, on account of the close relationship that exists between these disorders. Experiment proved that the effect of strychnine was increased by the addition of atropine in old and stubborn cases. One-fiftieth of a grain of strychnine and one one-hundred-and-fiftieth of a grain of atropine daily is the dose at first. This is increased gradually to one-twenty-fifth or one-twentieth of a grain of strychnine, and one one-hundredth of a grain of atropine. When a thorough impression is made, the drugs are administered every other day; and as the patient improves, they are gradually abandoned. Some cases get well by this means alone. Others require every measure that will overcome all physical abnormality. This would seem a wise precaution always, as one evidence of a general's skill would seem to be in the use of five cannon when he can command them, rather than trusting to luck and only one. The employment of strychnine and atropine hypodermically has also been of great service in the treatment of other forms of cough and dyspnœa.

ASTHMA CONSIDERED ESPECIALLY IN RELATION TO NASAL DISEASE.

This is the title of a new book by E. Schmiegelow, M.D., of Copenhagen. The author gives all due credit to Hock for bringing into general notice various considerations that had hitherto escaped attention; at the same time, this observer's exaggerated views are pointed out, and the following conclusions are given as the embodiment of experience and late research:

I. Asthma must be considered a bulbar neurosis.

II. This bulbar neurosis, which consists in an excessive reflex irritability of the respiratory centre, may be accompanied, though comparatively seldom, by a state of general nervousness; and in this case, as a rule, has the same etiological origin as hysteria or neurasthenia (whether inherited or acquired).

III. This bulbar neurosis may develop after weakening factors, such as childbirth, bleeding, continued fever, etc.

IV. This bulbar neurosis sometimes appears in otherwise apparently healthy individuals without any trace of other nervous phenomena, and in these cases it is presumably the result of frequent and strong irritations, which are

conducted to the respiratory centre from the nasal fibres of the trigeminus. To this irritation, the irritation of other nerves may also be added, such as that of laryngeal and pulmonary branches of the pneumogastric.

V. An asthmatic attack may originate in the mucous membrane of the nose, if only the necessary condition and the increased bulbar reflex-irritability be present; and irritations conducted to the medulla oblongata from any sensitive nerve whatever, are capable of causing an asthmatic attack.

VI. It is possible, in some cases, by suppression of peripheral irritation—as the careful treatment of a chronic nasal catarrh—to stop definitely the asthmatic attacks; but in many cases such treatment is only efficacious after general strengthening measures addressed to the central nervous system.

VII. In every case of asthma the nasal cavity should be examined, and the patient placed under special treatment whenever there is decided connection between the asthmatic attack and nasal difficulty.

VIII. Nasal disease may accidentally accompany cases of asthma, without having any etiological connection with the asthmatic attacks.

ASTHMA IN CHILDHOOD.

The "Progrès Médical," Jan. 25, 1890, has a notice of a monograph by Dr. Moncoro, on the asthma of childhood and its treatment. Etiology, as expressed in Brazil, does not include heredity to any great extent. Malaria, without causing the disease, contributes to the frequency of the attacks—which occur most often in spring and summer—and may aggravate the dyspnœa.

Hereditary syphilis was found in twenty-two out of forty-four cases. This enormous proportion is worthy of the most careful study. The asthma of childhood is benign, and makes for cure. Lobelia gives the best results. Five drops of pyrocin on a handkerchief, bound round the throat, is an excellent remedy to ward off attacks. The dose must be repeated four times a day. Morphine is *never* necessary. To prevent the recurrence of asthmatic attacks, tincture of iodine—four to ten drops a day—and iodide of sodium—one to four grains—have been most efficacious.

THE WAKEFULNESS OF NEURASTHENIA AS AFFECTED BY SEASIDE RESIDENCE: ITS SUCCESSFUL MANAGEMENT AND CURE.

In the "Medical News," April 5, 1890, Dr. W. H. Daley says, that in cases of insomnia which are made worse by

hypnotics, and especially by a residence at the seaside, quiet surroundings are essential, and when the tonic effect of the sea air proves beneficial the patient should be sent far enough inland where the roar of the sea cannot be heard. He also advocates a mild alterative and tonic treatment, with close attention to the state of the liver and stomach by means of mercurial laxatives. A quarter-grain calomel at bed-time with about two drachms fluid extract senna, as needed, is also recommended, until the yellow stools are tinged with bile. He allows stimulants in moderation when they are tolerated. In the congestive cases, Dr. Daley states that leeching the mastoid region will prove of nearly certain benefit, and very often cure.

HALLUCINATIONS FROM ATROPINE INSTILLATIONS IN THE EYE.

Dr. E. V. Belt, in the "Medical News," April 5, 1890, reports the case of a man where one drop atropine solution (4 grs. to 1 oz.) was instilled in the eye three times daily following an iridectomy. After the seventh instillation, he became delirious and had hallucinations. Such effects are extremely rare when the atropine is used only three times during the twenty-four hours.

LARGE DOSES OF KALI IODIDE.

Dr. B. M. Ricketts, in the "Cincinnati Medical Journal," March 15, 1890, states that the value of the iodides lies in the fact of their being tonic, and increasing the red blood-corpuscles. This, however, he believes is only the case in tertiary syphilis. If the amount of the drug eliminated by the kidneys is not in proportion to that administered, he advises the use of diuretics, especially large quantities of water, at frequent intervals. Two cases are recorded, one of which required 400 and the other 500 grains of this drug daily to ameliorate the symptoms of syphilis.

A CASE OF CENTRAL ABSCESS OF THE BRAIN, WITH SOME REMARKS UPON ITS RELATION TO CONTUSION.

In the "New York Medical Journal," March 29, 1890, Dr. Charles Phelps reports the case of a man who received a compound depressed fracture of the skull, just above and behind the right ear, from the falling of a brick. The depressed bone was removed and all went fairly well for twenty-four days, when he developed almost complete paralysis and anæsthesia of the left side, but not paralysis of the face. An incision was then made into the brain at

the right of fracture, and about two drachms of pus evacuated, but he died in 16 hours after the operation. At the autopsy an abscess cavity was found about half an inch to an inch below the cerebral surface at the seat of fracture, extending forward and inward about four centimetres from the surface, while its anterior posterior diameter was about two centimetres and a half, and its vertical scarcely more one centimetres. Around this cavity there was a wide area of softening. Dr. Phelps remarks that at no time in this case did the dura mater lose its integrity, and that the abscess had no topographical connection with the fracture. The paralysis and anæsthesia were evidently due to the cutting of communication by giving way of the softened fibres in the centrum ovale. He is inclined to regard central cerebral abscess as a result of contusion by *contre-coup*, and the later softening he believes may be caused by pyogenic germs which escape through the blood vessels and find their way to the contiguous tissue whose nutrition has been altered by the abscess pressure. He considers that if we are ever to recognize the early symptoms of contusion passing into abscess in time to be of any service to the patient, it must be by minute and methodical investigation, both before and after death, and extending over a great number of cases.

A CASE OF TRAUMATIC EPILEPSY ; OPERATION ; RECOVERY.

In the "New York Medical Journal," March 29, 1890, Dr. J. C. Reeve publishes the case of a boy, aged nine years, who was tramped upon by a horse, and received among other injuries a compound comminuted fracture of the right parietal bone. The loose fragments were removed and the patient remained well for two years, when he became epileptic. An operation was performed seven years later, November 20, 1888, which consisted in separating the scar tissue from the seat of the former fracture, an oblong space, where bone was entirely absent. He remained well till May 3, 1889, when three convulsions took place in one day, attended with fever, which attack was considered of malarial origin, but no convulsions have occurred since.

FRACTURE OF THE SKULL.

In a communication to the "Boston Medical and Surgical Journal," April 10, 1890, C. B. Porter, M. D., places on record the following histories :

CASE I.—Patient supposed to have been struck by a fast moving railroad train, sustained an extensive compound

comminuted fracture of skull on right side. The parts depressed were portions of the sphenoid, temporal, frontal and parietal bones. There was no paralysis. Elevation and removal of portions of the bones under strict antisepsis resulted in a speedy recovery.

CASE II.—Patient with a compound fractured skull presenting a depressed area one and a half inches by one and eighth inches in size, cracked through the middle and located to right of median line, and just behind the coronal suture. There was no paralysis, and after the fragments had been removed he made a good recovery in a month.

CASE III.—This patient received a pistol-shot wound of forehead (22 calibre) above eyebrow. There was temporary unconsciousness followed by aphasia and paralysis of right arm. Sensation not affected. (?) Non-interference was decided upon and in 24 days after the injury, he was practically well.

Dr. Porter attributes the success of these cases to the thorough antisepsis employed. In speaking of the treatment for simple comminuted depressed fractures, he rather condemns the practice of waiting for symptoms before operating, and says, "should not surgery in this dangerous class of cases take 'preventative trephining' as its motto, relying on modern antiseptic precautions to bring to a successful issue cases which had lead many times to worse than death?"

THREE CASES OF CEREBRAL TUMOR, WITH AUTOPSY.

By James J. Putnam, M. D., "Boston Medical and Surgical Journal," April 10, 1890.

CASE I.—Male, aged 39 years. Symptoma: Intense and incessant headache, almost exactly corresponding with situation of tumor; one well marked attack characterized by partial loss of consciousness and convulsive movements, limited to the left shoulder; double optic neuritis, much more intense on the side of the tumor. Subsequently the left arm and leg became weaker than the right. In six months the case terminated fatally, and at the autopsy a sarcomatous tumor was found involving the posterior half of the right middle frontal convolution. It measured two and a quarter by one and three-quarter inches, the longest diameter following the length of the convolution.

CASE II.—A gentleman, aged 58, without any history likely to have led to present illness. His symptoms were alteration of mental character, aphasia, partly sensory and partly motor, with ataxia; a general convulsion; epileptiform siezures, consisting of tremors in right arm and leg;

right hemiparesis of shifting intensity, terminating in complete paralysis with contraction; long-continued coma; Cheyne-Stokes respiration; death, with rapid elevation of temperature. The duration of his illness was about four months. The autopsy revealed two subcortical sarcomatous tumors, one occupying nearly the whole of the second left temporal convolution. The other was about the size of a horse-chestnut, and lay in the posterior part of the left parietal lobe.

CASE III.—Was that of a man 51 years old, who 22 years ago, was knocked from a car while passing under a bridge. He suffered from short attacks of arrest of power of speech for brief periods, without complete loss of consciousness, or of power of expression in writing, or of comprehension of simple speech or written signs, but preceded by trifling tremor of right hand; permanent, slight hesitancy in speech, with slight paraphasia; occasional general convulsions, without auro; frontal and occipital headache; mental failure; finally repeated attacks of slight convulsive action of flexors of right hand, with tendency to contracture, and paresis of extensors. It was thought the symptoms indicated a tumor just above the motor-speech area, and an operation was performed, which resulted fatally. After death the lesion was found to be situated in the supra-marginal gyrus, at its posterior end.

THE DETERMINATION OF THE DIASTASIC POWER OF EXTRACT OF MALT.

The "American Journal of Pharmacy," March, 1890, contains a method of estimating this power, as employed by R. A. Cripps, F. I. C. The process is as follows: 1. Prepare a mucilage by mixing one 1 grm. potato starch, dried at 212° F., with 10 c.c. of cold water; add 100 c.c. of boiling water, and boil the whole half an hour; allow to cool to 100° F., and make up the measure to 100 c.c. 2. Dissolve 5 grms. of the malt extract in water sufficient to produce 50 c.c. of solution. 3. Dissolve 1 grm. of iodine in 100 c.c. of water, by the aid of 2 grms. of iodide potassium. Fifty c.c. of starch solution is introduced into a flask, and kept at a temperature of 98° to 100° F., and 5 c.c. of the malt solution is added at the same temperature, gently mixed and at intervals of five minutes 4 c.c. of the liquid is poured into a test tube containing 1 c.c. of the iodine solution. A good malt extract gives no indication of starch or dextrine after 15 minutes, *i.e.*, it should digest its own weight of potato starch in 10 to 15 minutes at 98° to 100° F.

"American Journal of Pharmacy," April, 1890.—An item has lately appeared in medical journals calling attention to the danger of combining cherry-laurel water with morphine, as an insoluble precipitate of cyanide of morphine may be formed, and taken by the patient with the last portions of the mixture. John M. Maisch states that if the cherry-laurel water be made by distillation from the leaves, it cannot contain a cyanide, and the free hydrocyanic acid will not cause a precipitate, but if prepared by magnesia, it must contain magnesium cyanide, which would precipitate morphine.

"American Journal Pharmacy," April, 1890.—Geo. M. Beringer, Ph.G., considers that the process of preparing tincture of musk as directed by the U. S. Pharmacopœia is extravagant and wasteful. He suggests that in the pharmacopœial revision a strength of eight grains to the fluid ounce with diluted alcohol as a menstruum be adopted, which yields a tincture nearly identical in strength with that of the German Pharmacopœia.

MENTAL AND NERVOUS TROUBLES FOLLOWING GRIPPE.

According to the "*Révue générale de clinique et de thérapeutique*," March 26, 1890, Dr. Seglas as well as Dr. Huchard and Grasset, have observed the rekindling or aggravation of nervous and mental troubles by the recent epidemic in persons of neurotic disposition. Following a light attack of grippe appeared a trigeminal neuralgia that had given no trouble for ten years. The seizure lasted three weeks. An hysterical patient, who had been free from hysteria for two years, had an attack that lasted two days. Hysteria in another made its appearance following grippe after five years' absence. In neurasthenics, all the symptoms were greatly aggravated by the epidemic. Mental troubles following grippe are usually those of depression. Simple depression, intellectual and physical, absolute indifference, a sort of general sluggishness of all the mental faculties. Hallucinations were increased and classic mental conditions exaggerated by grippe.

FEVER IN EXOPHTHALMIC GOITRE.

The "*Progrès Médical*," Dec. 7, 1889, contains reference to the report of Gilles De La Tourette and H. Cathelineau on the study of fever in exophthalmic goitre. Basedow's disease without fever gives absolutely normal urine, even to normal phosphoric acid. When fever is present, spectroscopic examination reveals normal urea, phosphoric acid,

and urobilin. This fever, then, is unique, since the excreta possess none of the characteristics peculiar to other elevations of temperature. The authors now propose to extend their observations to hysterical fever, an extremely rare condition, no case having appeared at the Salpêtrière during the past two years.

L. F. B.

COMPOUND COMMINUTED FRACTURE OF THE SKULL;
EPILEPSY FOR FIVE YEARS; OPERATION;
RECOVERY.

The "Boston Medical and Surgical Journal," April 3, 1890, contains an account of this unusual case, by H. H. A. Beach, M. D. A girl when four years old was kicked in the head by a horse; had bleeding from the nose and left ear, and was unconscious for a week. Some pieces of bone were removed at the time of injury. About a month later, an abscess formed at the site of the wound, and on being opened, continued to discharge for six months. When nine years old she had full epileptic seizures, which continued to recur with increasing frequency and severity up to the time of operation. Examination showed a crucial cicatrix in the posterior temporal region, with a small depression where the lines crossed. Slight pressure here caused pain. After raising a flap, a nearly circular opening was disclosed in the skull, filled in with a web of cicatricial tissue, in the meshes of which there seemed to be a small cyst. The cicatrix was dissected from the brain and a sharp spicula of bone removed, which projected from the border of the opening. Since the operation the patient has improved mentally and physically, and more than nine months have now passed without a return of any epileptic symptoms.

BRIEF NOTES ON THE PROMPT RECOGNITION AND TREATMENT OF SYPHILIS OF THE BRAIN.

A short paper by J. Leonard Corning, M.A., M.D., contains the following:

The most important symptoms in cerebral syphilis are violent headaches, especially at night. Tender points on the scalp. Early attacks of vertigo and mental impairment, with inability to concentrate the thoughts or remember past events. Sensory and motor derangements, and also impairment of sight or speech. If spasms occur, they are usually limited to certain groups of muscles, and the same is generally true of paralysis. Both the monospasm and monoplegia tend to appear and disappear. Unlike the paralysis from apoplexy, this variety comes on gradually. The cra-

nial nerves are especially apt to be implicated. In treatment the iodides should be combined with mercury; the former to dissipate, the latter to prevent further agglomeration. ("New York Medical Journal," March 22, 1890).

ACUTE ASCENDING PARALYSIS FOLLOWED BY ATAXIC PARAPLEGIA.

A contribution to this subject is given by Sanger Brown, M. D. The etiology of the disease is obscure, but as an operating cause, exposure to cold ranks first. Among other causes may be mentioned convalescence from small-pox, typhoid fever and diphtheria. There are no constant premonitory symptoms, and the first definite sign of its approach is generally a weakness in the feet, which extends up the legs. From the leg the paralysis travels up the thigh, then to the muscles of the pelvis, loins, abdomen and thorax. The arms later become invaded, after which the diaphragm and neck muscles suffer, causing difficulty in deglutition and articulation. If the disease be not arrested, death from asphyxia occurs. Its duration varies from a few hours to four weeks, the usual period being two to four days. There is no fever, muscular wasting or changes in electro-excitability. No pathological lesions have been found. The prognosis is always grave till the march of symptoms is arrested. During the early stages the diagnosis from acute myelitis is impossible, and the treatment for this latter disorder is appropriate.

CASE.—R., age 42, male. Began to suffer with occipital headache in January, 1887, and on June 9th, 1887, noticed slight numbness of the toes of left foot. After this, numbness and weakness extended up both limbs and reached to the umbilicus. It then appeared in the fingers of each hand, but here its progress ceased; the duration of the attack being about forty-eight hours. Six weeks later the power of movement began to return, and in two months he could stand supported. Incoördination was noticed a few months after he began to walk. During the past six months distinct improvement has occurred. Present condition: Well nourished and general health good. The gait is typical of ataxic paraplegia. Myotatic irritability is exaggerated and sensation is blunted for touch and temperature. The left pupil is a trifle dilated. There seems to be a strong support to Gower's hypothesis, viz., that this disease is due to a toxic or other influence acting on the filamentous terminations of the upper segment in the gray matter of the cord. ("Medical Record," March 22, 1890).

METHODS OF EXAMINATION IN MEDICO-LEGAL CASES
INVOLVING SUITS FOR DAMAGES.

In a reprint from the "Boston Medical and Surgical Journal," Dec. 19 and 26, 1890, P. C. Knapp, A. M., M. D., gives a number of valuable directions to guide the physician in making thorough examinations in this class of cases. He avoids saying much of methods requiring complicated apparatus, and mostly confines his remarks to those means in examining special organs, like the eye and ear, which the general practitioner ought to understand. The article contains the following headings, under each of which Dr. Knapp gives many useful hints on the subject: Psychical and Motor Symptoms; Reflexes; Special Senses; Thoracic, Abdominal and Genito-Urinary Symptoms. It is a very readable pamphlet and well worth perusing.

INFANTILE CONVULSIONS.

A protest is offered by W. A. Dickey, M. D., against the routine treatment of giving hot baths to children in convulsions. He advises the employment of the thermometer in all these cases before beginning treatment. Where the temperature is found to be elevated, 104 or more, a cool bath is indicated, but a hot bath should never be used under these circumstances, as he considers that it only adds fuel to the flames. (The "Medical Compend.," Feb., 1890).

A. F.

ANTIPYRINE IN MENTAL ALIENATION.

Dr. Raffaele Roscioli ("Il Manicomo," No. 3, 1889). The drug was administered in varying doses, and both internally and hypodermically, to fourteen patients, four with melancholia, two actively maniacal, one paralytic, one hysterical, one with the delusion of persecution, and five epileptics.

In mania, antipyrine has no effect either as a sedative to the nervous system or as a hypnotic. In melancholia and general paralysis, the effect is equally negative; but with large doses there would be sometimes an aggravation of the symptoms. In hysterical mania, with the morphine habit, there was no result either in the psycho-pathia or the morphine habit. The effects were positive and rapid in the convulsive seizures of epilepsy, but none in the pre- and post-epileptic, which declined, by comparison with

the rest, during the cure; or, in grave and inveterate cases, diminished in number and intensity as soon as the antipyrine was discontinued.

In comparing the use of antipyrine with bromide, in epilepsy, the writer thinks the action of the former is much more rapid, but less enduring. The long-continued use of bromide undoubtedly leaves the patient in a state of mental torpor very nearly followed by dementia, while the torpor produced by antipyrine vanishes without grave consequences to the intelligence.

Sée has asserted that antipyrine can be substituted entirely for morphine in the cases which have symptoms of a neuralgic nature. In the case that the author tried antipyrine, it was powerless to allay the intense neuralgia which afflicted the patient.

Dr. Roscioli thinks that the weight of the body is probably diminished by the use of antipyrine. The temperature in general was not influenced; it remained normal. The pulse was generally strong and rhythmical.

The following are abstracts given in the "Report of the Sixth Congresso Freniatico Italiano," and published in the "Rivista Sperimentale di Freniatria e di Medicina Legale," vol. xvi., fasc. i.-ii.:

I. CANGER.—HYPODERMIC INJECTIONS OF THE AQUEOUS EXTRACT OF OPIUM IN MENTAL DISEASES.

The author communicates twenty-seven observations of patients treated in this manner, and comes to the conclusion that the injections of this preparation are better tolerated than any alkaloid of opium. It helped greatly in the depressive form, especially in the anxious, less in states of exalted mania, but in violent mania it was harmful.

It succeeded in a single case of hysteria in which it was tried. The injection calms the motor disturbance in the forms of acute sensorial insanity, and in all those psychoses acts as a general tonic to the central nervous system, increasing the nutrition.

II. AGOSTINI AND BERARDUCCI.—ANTIPYRINE AS A SEDATIVE IN THE INSANE.

The authors, who take the ground that much of the agitation of the insane is determined by painful sensations, experimented with eight cases, two of declared mania (psycho-neuroses) and six with maniacal excitement, symptomatic of cerebral anatomical lesions (partial meningitis, etc.). In the psycho-neurotic the antipyrine gave no relief.

In the other cases, in doses of one to two grammes, it gave a calm of sufficient duration. They propose to experiment further with the remedy.

III. AMADEI.—SUPPRESSION OF THE EPILEPTIC ATTACK.

The author said that antipyrine succeeded only in cases where the disturbance was slight; that the bromide was ineffectual in severe cases. It had been shown that the two drugs combined gave good results oftentimes in obstinate cases. Using this means, there had been no epileptic attacks for four months in the Asylum at Cremona. It was a little premature to announce these results; but to make the communication might insure greater experimentation.

NATURAL AND SIMULATED SOMNAMBULISM.

Dr. Catrin, of the Military School at Lyons, wrote an article in the "Lyons Medical," upon which Dr. Filippi comments in "Lo Sperimentale" (December, 1889).

Dr. Catrin had a case of simulated somnambulism, and wished to suppress, if possible, this most disorderly malinger among the troops. He quotes from Charcot the signs of natural somnambulism, as follows:

1. Natural somnambulism is a disorder of childhood and of early youth.
2. It is a disease rare in the adult.
3. It is produced almost always at the same hour of the morning or in the middle of the night.
4. The open eyes are a symptom of the first importance.
5. The true somnambulist pays no attention whatever to anybody who would try to impede his progress.
6. It is a rare disease, especially in the non-hysterical.

According to Dr. Catrin, all those cases which do not present the individual conditions and the clinical phenomena of natural somnambulism should be suspected of simulated somnambulism. But Dr. Filippi has his doubts as to the propriety of these conclusions, although he recognizes, at the same time, the merit of the work and of this special study in military medicine.

REFLEX EPILEPSY FROM CATARRH AND DILATATION OF THE STOMACH.

Dr. Luigi Zacchi ("Lo Sperimentale," January, 1890). The author narrates the case of a young man, twenty-four years old, who had an epileptic seizure, judged to be such from the fact that it was preceded by a feeling of malaise

(the aura) and by a cry, and that he had tonic and clonic convulsions, foaming at the mouth, wounding of the tongue, insensibility to strong pricks, passing of the urine, and the characteristic aspect.

That the epilepsy depended upon the pathological condition of the stomach was shown by the cessation of the seizures from the time the stomach began to function regularly. The writer draws the attention to the absolute inefficacy of bromides to combat gastric epilepsy or to modify it; and likewise to the inefficacy of remedies for the catarrh and dilatation of the stomach, which yielded readily to repeated washings, which removed the accumulated secretions and stimulated to contractions the atonic and torpid muscular structure.

To sustain his diagnosis and the points that he makes, Dr. Zacchi cites a case of a woman whom he once saw with reflex nervous symptoms, face drawn and bathed with cold perspiration, eyes wide open and without expression, pulse slow and weak, unable to make an intelligent answer. She indicated with her hand that the stomach was the seat of her principal trouble. She recovered after she was made to vomit and had ejected a large amount of undigested food.

He also mentions a transitory reflex aphasia, the result of indigestion. G. P.

Society Reports.

AMERICAN NEUROLOGICAL ASSOCIATION.

*Sixteenth Annual Meeting, held at Philadelphia, June 4th,
5th, and 6th, 1890.*

(CONTINUED.)

Dr. PETERSON then read a paper, entitled: "A Case of Locomotor Ataxia associated with Nuclear Cranial Nerve Palsies and with Muscular Atrophies." (See page 450.)

Dr. C. K. MILLS thought that the case might be some peculiar irregular form of syringo-myelia.

Dr. E. D. FISHER suggested that the case was one of amyotrophic lateral sclerosis.

Dr. SACHS thought the case closely resembled one of his, in which the diagnosis was polio-myelitis, although there were more symptoms of tabes dorsalis than in his case.

Dr. PETERSON did not think the case one of syringomyelia, owing to the very symmetrical distribution of the nuclear palsies, and because there was no anæsthesia of the upper extremities, face or trunk. There was no symptom of amyotrophic lateral sclerosis except the atrophy of certain muscles, which was subsequent to the disappearance of the knee-jerk. The speaker thought the case was probably one of extreme locomotor ataxia, with nuclear cranial nerve palsies and muscular atrophies dependent upon peripheral neuritis, as described by Déjerine.

A CASE OF COMPLETE PARAPLEGIA CURED BY OPERATION.

Dr. F. X. DERCUM presented a middle-aged man with the following history, in brief: The patient had, in 1887, suffered severe pain in the arms and shoulders. The pain had appeared to be referred to the principal nerve tracts. Soon after this he had lost power in his legs. Then about October of 1888, in addition to complete paraplegia, there was loss of sensibility, but with constricting pains about the upper portion of the chest. There was also complete paralysis of both sphincters. Examination had revealed the fact that the man's back was very painful over the third, fourth and fifth dorsal vertebræ. Thinking there might possibly be some local cause for the paralysis, the speaker had called others in consultation. It was agreed that the case was one in which trephining should be tried. The spines and arches of the first to the fifth dorsal vertebræ, inclusive, were accordingly removed. The dura was found somewhat abnormally resistant to the touch, and was opened. Adhesions existed between the dura and pia. After the operation, which the man had borne well, he had at once said that his pain was absent. A few days afterwards he was able to feel at the foot, when touched. Then he was sensible that his hands were cold, and was able in a few days more to move his toes. There had been a very gradual but steady progress towards complete recovery. He had also regained control of the sphincters. Whether the result was to be ascribed to local relief from pressure, or to reaction from the shock of the surgical operation, the speaker did not venture to suggest. The paraplegia was probably the result partly of pressure and partly of myelitis.

The President, in commenting on this unique case, characterized it as almost unprecedented.

Dr. PUTNAM said the case seemed to be one of unusual interest, and thought that they must all welcome such an

advance in the surgery of this region. In a case of his, tumor of the spine was diagnosed, but operation was rejected. The rapidity of the development had suggested cancer of the vertebræ. He did not believe it was possible to tell from the severity of the symptoms, or the rapidity of their development, whether the conditions present were such as would contraindicate operation, and it ought to be done, even if the patient was in a pretty bad way, as a matter of investigation. He would call attention to the myelitis of Pott's disease and the paralysis due to œdema. In the case he had mentioned, the disease had turned out to be cancer. Horsely had reported among his cures one in which the disease had come on rapidly, and in which the local affection was carcinoma, but situated posteriorly. The case they had just seen would give a great deal of encouragement for the recommendation of such means of treatment.

Dr. V. P. GIBNEY said he was extremely delighted with the brilliant results in this case. From the way the patient now held his head he should be inclined to look upon the case as an old Pott's disease, and that the thickening found was the result of an inflammatory process within the bodies of the vertebræ. There might have been pressure myelitis from thickening around the vertebræ. The case was somewhat remarkable, considering the age of the patient. Surgeons had been chary of operating in these cases, though the operation was comparatively an easy one, at least simple in detail, and with the antiseptic and aseptic methods of to-day, it was practically impossible to have bad results. Recovery was what ought to be expected. Most of the cases so far operated upon, however, were of traumatic origin, old dislocations, fractures, and so forth. Of the cases of pressure myelitis in children a great many got well without treatment, while a certain number resisted all forms of intemperance. He had had a girl under treatment, by traction, for twelve months, and she had made a complete recovery. Still if it was practicable to cut down and do a laminectomy and get at the radical trouble, it would be much better.

The President said they would be glad to know to what extent the present position of the head corresponded with the position before the operation.

Dr. DERCUM said the position was then normal. He considered the present inclination of the head forward as due to loss of certain attachments of the trapezius muscles at their points of insertion into the spinous processes. It

should be stated that there might have been a rheumatic element in this case affecting the meninges of the cord. The patient had described his pains as rheumatic, but they had subsided after the operation.

The President said he would like to ask Dr. Gibney as to the deformity in which there was extreme bending forward of the neck.

Dr. GIBNEY said these conditions sometimes obtained to an indefinite extent. It might be a senile hyphosis. Though it would perhaps be urged that Pott's disease at such an age was impossible, yet, as a matter of fact, there were quite a number in whom it developed after fifty or sixty, following a fall or injury. The position of this man's head might be due to loss of substance of the bodies of the vertebræ.

Dr. W. M. BULLARD thought the rapid advance in spinal surgery should lead to more definite conclusions as to the cases of Pott's disease suitable for operation. He was inclined to agree with Dr. Gibney that the case before them might be one of this nature. He did not think it necessary to avoid operation on account of the rapid development of symptoms. Extreme care should be observed in operations for Pott's disease in children. Many of these cases did well eventually if suitably treated by rest, and so on.

Dr. SACHS said he was gratified to hear the report of such a case as Dr. Dercum had presented. It went to show that many of the cases hitherto operated upon had been badly selected. It was difficult as a rule to tell whether the operation promised well or not. When cases could be selected statistics would probably be more encouraging and favorable.

The President thought that the theory of rheumatism in this case could not be entirely disposed of.

Dr. C. L. DANA said he did not share the optimistic views which Dr. Putnam had advanced, and should be sorry to start a boom in spinal surgery. If the history of all the operations already performed in this field was known, he thought they would all be disposed to follow the conservative suggestions of Dr. Sachs, and would only deal with selected cases. The operation was not, in the speaker's opinion, a simple one.

Dr. GIBNEY said that he for one did not by any means desire to start a boom in spinal surgery upon the results of a single cure. What he had meant by an easy operation was that in these old cases of Pott's disease the spinous processes were more prominent, and the soft parts were thin and readily got at. Cases should, of course, be most

carefully selected; but when patients were evidently going down and the cases began to assume a serious aspect, he thought that surgical interference, properly undertaken, was justifiable.

Dr. DERCUM thought that the proper cases were difficult of selection, and it was only now and then that success could be hoped for. He regarded the result in his case as fortuitous.

Dr. G. M. HAMMOND presented a report on the "Pathological Findings in the Original Case of Athetosis," on which Dr. W. A. Hammond's description of athetosis was based. After briefly referring to the case, Dr. Hammond stated that the portion involved in the lesion consisted of fibrous connective tissue. Topographically, the lesion was a lengthy one in the antero-posterior direction, parallel in its short axis with the internal capsule. Its posterior end invaded the stratum zonale of the thalamus in its posterior third, and the posterior third of the posterior half of the internal capsule. In its anterior extension it crossed the capsule, invading the posterior third of the outer articulus of the lenticular nucleus. The author called attention to the fact that the motor tract was not implicated in the lesion, and claimed that this case was further evidence of his theory that athetosis was caused by irritation of the thalamus, the striatum, or the cortex, and not by a lesion of the motor tract.

Dr. E. C. SPITZKA reported a case of hemi-athetosis, in which the lesion was found to be in the same situation as the one in Dr. Hammond's case.

Dr. E. C. SEGUIN presented a paper on "Atheto-Choreic Spasm of the Right Side of the Body." The post-mortem showed a glioma of the left thalamus opticus and adjacent internal capsule.

Dr. Seguin's views were that all cases of athetoid and choreiform movements following hemiplegia were due to lesions involving the thalamus and adjacent capsule.

Dr. PUTNAM then read a paper on "A Form of Poly-Neuritis, Probably Analogous to or Identical with Beri-Beri Occurring in Sea-Faring Men in Northern Latitudes." (See page 495.)

Dr. GRAY asked if these ships had carried large quantities of ice, because he had seen cases of neuritis following the handling of ice, which had promptly subsided if this cause was removed. The condition might in Dr. Putnam's cases have so originated and then become infectious or contagious.

Dr. BIRDSALL inquired as to the amount of air supplied to the hold of these vessels, and to the seamen's quarters. Cases recently reported to him had led to the impression that the men who had died had been specially exposed to bad air in the hold, while those in good quarters had escaped. He agreed, also, that cold was an important element in the production of all forms of neuritis.

Dr. HERTER asked whether the men's diet had been of fish.

Dr. PUTNAM said that fish had not been the diet, but salt pork, sometimes fresh vegetables and fresh meat. But in one of the worst cases it had been salt pork.

Dr. BULLARD, of Boston, then read a paper on

DIFFUSE CORTICAL SCLEROSIS OF THE BRAIN IN CHILDREN.

Under this title he places those cases of diffuse cerebral sclerosis in which the cortical layers of the cerebrum are primarily or principally affected and where the microscope shows the presence in these layers of a homogeneous ground-substance or of a finely fibrous network with a few spider-cells in the meshes occupying the position of the neuroglia.

Accurate accounts of such cases with autopsies, in children, are rare. Doubtful whether cases of cortical sclerosis can always be distinguished from forms of diffuse sclerosis, in which the cortical layers are not specially affected. "From these we have not yet the evidence to determine in how far our cases differ."

Cases analogous reported:

Personal.—Boy, twelve years old; family history negative; always healthy; was bright, intelligent, did well at school. In August, 1886, had a fall, striking his head against curbstone and making wound over right parietal bone, of which the scar still remains. Wound suppurated, not painful, but some sensitiveness over head all winter. Three weeks after injury he forgot the baby, whom he had taken out-of-doors. (He was always fond of him, and was accustomed to take care of him.) Two months later he spent the whole night out, because he could not find his way home nor tell where he lived.

April, 1887.—Gradual loss of memory since accident; loss of interest in objects about him. Clonic spasm of tongue now ceased; clonic spasm of left face and arm. Has become awkward in use of extremities.

Present Condition.—Well developed and nourished; very forgetful and childish; markedly *weak-minded*. Sits much of the time with vacant expression; understands fairly what is said to him. Likes to sit by window and watch what goes by. Can read the name on a wagon, but attaches no meaning to the words. Speech slow, indistinct; occasionally omits words. Head of normal shape and size. Scar one and a half inch long over right parietal bone. Probable paresis of all extremities. Slight, but marked, incoordination, especially left. Sensation normal; knee-jerks exaggerated; waddles in walking. Double neuro-retinitis.

Consultation with Dr. Russell.

Operation.—Trephining over cicatrice in right parietal region. Button of bone replaced. (Specimen shown.)

June, 1887 (ten months after the fall).—After operation, clonic spasms ceased temporarily, then gradually increased and involved all extremities. Aphasia; screams much. Is now almost helpless. Lies on mattress on floor; cannot get up alone, but can walk a little with assistance.

August, 1887 (twelve months).—Has had paralysis of sphincters for some time. Cannot speak; cannot sit up; cannot use hands or feet much. Morbid dementia; recognizes mother; inclined to pull things to pieces; grinds teeth.

November, 1887.—Left extremities almost totally paralyzed. Transferred to Boston Lunatic Hospital. Died in condition of extreme dementia (fifteen months after the accident).

Autopsy (made by Dr. Gannett).—Œdema of pia; chronic lepto-meningitis; atrophy of brain; chronic internal hydrocephalus, secondary to atrophy of brain; chronic ependymitis of fourth ventricle; no evidence of compression or obstruction of venæ Galeni. Brain did not quite fill cavity of cranium; weighed 1,260 grammes. Pia showed slight thickening and opacity; nowhere adherent.

Microscopic Examination.—Pia showed a slight degree of thickening from growth of fibrous tissue. Brain: First layer of cortex showed the finely fibrous network with a few spider-cells in the mesh, due to atrophy of the nerve-fibres and an increase in the neuroglia. There was a slight degree of nerve-cell infiltration of the adventitial sheaths of the blood-vessels in the cortex. Beyond these nothing abnormal was observed.

The clinical picture presented (Moore's case and my own) is as follows: A varying time after a fall, intellectual impairment—at first simply obtuseness, general dullness,

and loss of memory; then clonic spasms of face and extremities, which may later be followed by epileptic seizures. Loss of motor power, sometimes appearing suddenly as a hemiplegia, sometimes very gradually, but usually involving the extremities, on one side especially; sometimes inco-ordination of extremities with similar distribution. Speech affected, slow, thick, or aphasic, accompanying a right hemiplegia. Loss of power in extremities progressive, in no case absolute; involvement of sphincters. As the disease progresses, the spasmodic manifestations tend to increase in frequency and severity; epileptic attacks and opisthotonus may occur. Neuro-retinitis or optic neuritis. Sensory symptoms almost absent.

Progressive Failure of Intellect.—Simple rapidly progressive deterioration of all the mental powers, a pure dementia uncomplicated by intervals of excitement or violence. No hallucinations or delusions detected.

"The symptoms of this affection are those of a gradually progressive mental deterioration, uncomplicated by hallucinatory or delusional conditions and a gradually progressive loss of power, involving all the extremities, the trunk-muscles, and the sphincters, loss of sight, loss of speech—all these accompanied by symptoms of motor explosion, clonic spasms, tonic spasms, and eventually epileptic seizures." Inco-ordination; absence of sensory symptoms. Neither tremor nor nystagmus in either of these cases.

Diagnosis.—The most important practical point is to determine whether we are dealing with a diffuse process or a local one. In certain cases this is by no means easy to determine. Where the explosive symptoms predominate and the paresis is slight or unilateral, and especially where, as in my case, a cicatrix exists over the motor region on the side opposite to that of the hemiparesis, the possibility of a localized starting-point cannot be excluded, although the marked mental enfeeblement, in conjunction with the motor symptoms, would point to an affection of considerable extent, and would suggest the existence of a subacute or chronic form of lepto-meningitis. 'It is the possibility of a localized lesion or new growth, accompanied by a chronic or subacute inflammation of the meningeal membranes, which may, at least for a time, render an absolute diagnosis impossible. "A cerebral tumor, uncomplicated by meningitis, would be very unlikely to produce this combination of symptoms."

Differential diagnosis from multiple sclerosis; from congenital and early forms of cerebral atrophy; from all forms of lobar or partial atrophy; from primary hydrocephalus.

Provisional Conclusions.—There exists in children a non-congenital form of diffuse cerebral sclerosis in which the cortical layers of the brain are more specially affected, and which is distinguished from the other forms by its appearance in healthy children, either without known cause or after traumata, by the steadily progressive character of its symptoms and by the especial prominence of the gradually increasing *dementia*, which finally reaches an extreme degree without a corresponding loss of motor power, and while the sensation is comparatively unaffected.

[TO BE CONTINUED.]

NEW YORK NEUROLOGICAL SOCIETY.

Stated Meeting, May 6, 1890.

THE President, DR. LANDON CARTER GRAY, in the chair.

Dr. LANDON CARTER GRAY read a paper, entitled: "Can We Diagnose Hyperæmia and Anæmia of the Brain and Cord?" After citing the views of such recent writers as Gowers, Strümpell; Liebermeister, Ross, Seeligmüller and Hammond, and showing that they all, with the exception of the one last named, spoke with considerable skepticism of the possibility of diagnosis, the writer then passed in review the symptoms upon which the diagnosis is supposed to be made. Of these cephalgia, flushing and pallor of the face, vertigo, tinnitus aurium, insomnia, delirium, were, he said, general symptoms which can only derive diagnostic value from their associated symptoms. Slight retinal changes of vascularity are almost impossible to differentiate from the normal, as every well-informed ophthalmologist knows, and more marked retinal changes, such as neuro-retinitis, are indicative of organic intracranial disease. Myosis and motor and sensory paralysis have never yet been shown to have been caused by simple hyperæmia or anæmia of the brain. If the symptoms of apoplexy, paralysis, convulsions, a soporific condition, mania and aphasia, which Dr. Hammond states to be indicative of cerebral hyperæmia and anæmia are symptoms of organic intracranial disease, and the pathological data which are given by Dr. Hammond, such as enlarged and dilated blood-vessels, increase in consistence and density of the white matter, red or violet hue of the gray matter, subarachnoid or ventricular or choroid effusion, hematine, the *l'état criblé*

of Calmeil, miliary aneurisms, certainly show the existence of such organic disease as pronounced as in many cases of general paresis of the insane or in the severer cases of chorea or certain cases of intracranial syphilis. There is no proof that spinal anæmia or hyperæmia can be diagnosed whilst the symptoms given by many authors, on the one hand, are either too vague for differentiation from many slight affections, or, on the other hand, that of actual organic myelitis. Many diseases in the past, when they were poorly understood, were thought to be due to hyperæmia or anæmia of the brain or cord, such as meningitis, general paresis, porencephalitis, cerebral and spinal syphilis, disseminated sclerosis, disease of the medulla oblongata, myelitis of the anterior horns of the spinal cord, central and transverse myelitis, syringo-myelitis, progressive muscular atrophy, acute ascending or Landry's paralysis, locomotor ataxia, especially in its early stages, many forms of neuritis, focal disease of the brain in the light of our modern knowledge of localization, lateral sclerosis, many forms of lithæmia and neurasthenia. For these reasons the author does not believe that hyperæmia or anæmia of the brain or cord can be diagnosticated by means of the symptoms alone. If there are evidences of some intracranial disturbance, such as headache, delirium, vertigo, tinnitus aurium, insomnia, flushing or pallor of the face, and with these some concomitant conditions, making it reasonable to suppose that there might exist congestion or anæmia of the brain or cord, such a diagnosis might then be probable. Thus certain cardiac conditions, lesions of the great vessels of the chest and abdomen and thorax, certain forms of hepatic disease, tumors making pressure upon these great vessels, leucocythemia, intense mental complications or exposure to great heat; these conditions associated with the general symptoms might make it probable that the general symptoms were due to hyperæmia or anæmia, as the case might be, of the brain, but the writer does not believe that there are any pathological observations on record that would warrant a diagnosis of spinal anæmia or hyperæmia.

DISCUSSION.

Dr. W. R. BIRDSALL said he must admit that he had reached about the same conclusions as the reader of the address; that it was next to impossible to determine between the conditions of hyperæmia and anæmia by the nervous symptoms alone. There were, however, times when it was undoubtedly possible to recognize these states

from the general condition of the patient's system and from the course and character of the disease. Still, he thought there existed a great deal of confusion as to how the terms should be used. For instance, the term anæmia was often employed to express a mere malnutrition of the part. This application would be inappropriate in reference to the sense in which the President had discussed the subject. If it were a question whether the organs were supplied with too much or too little blood, of course variations in the quality of the blood must make considerable difference in the resulting symptoms, but he failed to appreciate how such symptoms could throw any light on the question. He believed that a large part of the symptoms which were brought forward as evidence of hyperæmia and anæmia were really conditions of disease due to malnutrition of the parts from the presence of poisonous substances in the blood, and not necessarily evidence of a defective or excessive supply of blood.

Dr. W. M. LESZYNSKY said that some years ago it had been the rule to diagnosticate cerebral hyperæmia upon the lines laid down by Dr. Hammond. The correctness of the diagnoses so made was assumed from the results of the results of the treatment. Patients in whom such symptoms were supposed to be developing were treated with bromides and ergot, because these drugs were thought to act directly upon the blood-vessels and the cerebral circulation. Many patients were benefited, and the diagnosis was therefore considered to be correct. The conclusion had, however, not been sustained. Investigation had demonstrated also that the condition of the retinal blood-vessels could not be taken as wholly indicative of the state of the circulation in the brain.

Dr. MARY PUTNAM JACOBI remarked that many of the clinical symptoms which were most nearly associated with anæmia of the brain and which implied malnutrition of the cortex were unattended by any symptoms of general anæmia. The question of anæmia, therefore, so far as the brain was concerned, was complicated by the possibility of there being simply a vaso-motor spasm in the brain, which would diminish the blood supply and yet give no impression of general anæmia.

Dr. M. ALLEN STARR expressed his hearty agreement with the statements and conclusions of the writer of the paper. He was perfectly satisfied as to the impossibility of diagnosing between cerebral anæmia and hyperæmia. To pretend to do this was merely an evidence of incompetency on the part of the physician.

Books, Pamphlets, etc. Received.

BOOKS RECEIVED.

- THE ANATOMY OF THE CENTRAL NERVOUS ORGANS: In Health and Disease. By Dr. Heinrich Obersteiner. Translated by Alix Hill, M.A., M.D., M.R.C.S. Illustrated. Philadelphia: P. Blakiston, Son & Co.
- FLUSHING AND MORBID BLUSHING: their Pathology and Treatment. By Harry Campbell, M.D., Bs. (Lond.). London, 1890: H. K. Lewis.
- SPINAL IRRITATION, and Some Points in the Diagnosis of Affections that may be Mistaken for It. By J. K. Eskridge, M.D. Reprint.
- THE TREATMENT OF POST-DISPLACEMENTS OF THE UTERUS. By H. J. Boldt, M.D. Reprint.
- ABSCESS OF THE BRAIN: Operation; Death on the Ninth Day. By J. T. Eskridge, M.D. Reprint.
-

PAMPHLETS, ETC., RECEIVED.

- THE ARREST AND PARTIAL RESORPTION OF IMMATURE CATARACT, WITH RESTORATION OF READING-POWER. By Richard Kalish, A.M., M.D. Reprint.
- HEREDITARY CHOREA: With a Contribution of Eight Additional Cases of the Disease. By C. M. Hay, M.D. Reprint.
- A CASE OF PARANOIA: Illustrating the Features of Imperative Conceptions. By Theodore Diller, M.D. Reprint.
- CIRCULAR INSANITY. By Theodore Diller, M.D. Reprint.
- SOME POINTS ON THE PERINÆUM AND FORCEPS. By T. J. McGillicuddy, M.D. Reprint.
- SALPINGO: OOPHORECTOMY AND ITS RESULTS. By H. J. Boldt, M.D. Reprint.
- THE PERONEAL FORM OR LEG TYPE OF PROGRESSIVE MUSCULAR ATROPHY. By B. Sachs, M.D. Reprint.
- 73D ANNUAL REPORT (1890) FRIENDS' ASYLUM FOR THE INSANE.
- 66TH ANNUAL REPORT (1890) RETREAT FOR THE INSANE, Hartford, Conn.
- 7TH ANNUAL REPORT (1890) McLEAN ASYLUM TRAINING-SCHOOL FOR NURSES.
- 76TH ANNUAL REPORT MASSACHUSETTS GENERAL HOSPITAL AND McLEAN ASYLUM.

THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

PROGRESSIVE SENILE PARAPLEGIA: A CONTRIBUTION TO THE STUDY OF NON-INFLAMMATORY SOFTENING OF THE CORD.¹

By CHARLES L. DANA, M.D.,

Professor of Nervous Diseases, N. N. Post-Graduate College.

THE literature of the senile cord is quite meagre. Cruveilhier, Geist, Rokitansky, Durand-Fardel, Empes, are the principal older writers upon the subject. Leyden, in his text-book, has given the most complete description of spinal senile changes.

He shows that there is a tendency for the cord to atrophy slightly and become harder. He finds amyloid bodies very frequently around the central canal and periphery; the cells in the gray matter are somewhat wasted and pigmented, the vessel-walls are thickened and show hyaline degeneration. There seems to be a tendency to stasis of the blood, and foci of arterial thrombosis are found.

The symptoms associated with these changes are: senile tremor, progressive senile weakness and paraplegia, and acute or subacute paralyses from focal softening.

Progressive senile paraplegia in its fully developed form seems to be quite rare. Empes, in 1862 ("Arch. gén. de

¹ Read at the Annual Meeting of the American Neurological Association, June, 1890.

Médecine"), described what he calls a "progressive senile enfeeblement," which amounted eventually to paraplegia. The exact pathology of this condition has not been firmly established.

The case which I have to report is important, in that it gives evidence of what the pathological condition in these cases is.

It throws some light also on the subject of non-inflammatory softening of the cord.

Systematic writers have generally confessed themselves in the dark as regards the existence of non-inflammatory softening of the cord; and in Gowers' work no mention is made of this subject.

Acute softening has been described usually as synonymous with acute myelitis; but the term is wrongly used, and should not be applied to inflammatory processes at all. Of late it has been suggested that some of the cases of acute myelitis are in fact primarily necrotic processes; but not much evidence has accumulated upon the point.

At all events, the process which I am to describe does not belong to the acute class, but is one strictly of a chronic myelomalacia, and one of a systemic character.

The patient was a man, married, born in Bohemia, seventy years of age; very little known of his early life, except that he was married and had healthy children. About four years ago, at the age of sixty-five or sixty-six, he noticed some weakness in the legs, but he had no pain. This weakness progressed until a year ago, when he was no longer able to walk. About this time he began to have some incontinence of the urine and trouble with the rectal sphincters.

His disease progressed until he entered the Montefiori Home in 1889.

Status Præsens.—The patient presents the appearance of senility. He lies in bed with his legs drawn up and nearly immovable. His head is drawn a little to the left. The movements of his arms, face, eyes, and tongue are normal, nor are there any trophic or sensory disturbances here. The disease, except for the slight contracture of the neck, is limited to the lower extremities. These are much wasted and contracted. The knee-jerks are gone, nor is there any clonus or trepidation.

Sensation is normal everywhere; there being, if anything, hyperæsthesia. No pains in the leg or girdle-pains or bed-sores. The urine (which is albuminous) dribbles from the bladder, and there is incontinence of fæces.

The patient's mind was clear, but showed evidences of senility.

The symptoms changed very little, and the patient died finally of exhaustion.



Autopsy was made by Dr. Leo Ettinger, house-physician.
Brain not examined.

The Spinal Cord.—On opening the spinal canal, no apparent abnormalities were observed in the meninges. The cord itself, externally, showed no notable abnormalities to the naked eye. Sections of the fresh cord showed changes in the anterior horns in its lower portion.

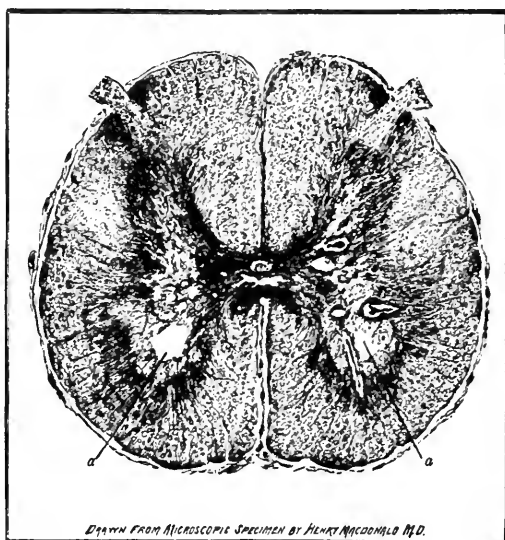
The cord was hardened in Müller's fluid, and stained with hæmatoxylin (Weigert's) and aniline blue.

Microscopical Examination.—There was considerable thickening of the pia-mater. Throughout the cord there was seen evidence of marked arterial sclerosis, with thickening of the walls and reduction of cavity, particularly of the smaller vessels.

These changes were most pronounced in the vessels of the anterior median fissure and their branches and in the peripheral arteries radiating toward the anterior horns.

Throughout the cord there was atrophy and absence of the cells of the anterior horns and intermediate gray. In the lumbar region the anterior horns had dropped out, leaving only a ring of healthy gray matter. There was no secondary connective-tissue proliferation and no shrinking of substance.

In the lower dorsal region the anterior horns were reduced to an amorphous granular material thickly interspersed with distended capillaries and here and there a dilated vessel with thickened walls. Ascending the cord,



DIAPYCN FROM MICROSCOPIC SPECIMEN BY HENRY MACDONALD M.D.

the anterior horns showed more cells, especially upon the periphery, in parts supplied by the peripheral arteries of the pia. In other words, the cerebral arteries were most affected.

The arterial sclerosis affected also the posterior cornua, but to a less extent, and nowhere was there loss of substance.

There was a very noticeable sclerosis in the pyramidal tract on both sides, but more marked high up in the cervical region.

In other respects the white columns were normal. There was loss of fibres in the anterior roots, but not in the posterior.

The anterior cornual cells could be seen in all stages of atrophy, but without any vacuolation or much pigmentation.

The columns of Clark were slightly affected.

The condition, in brief, was one of degenerative endarteritis with sclerosis, obliteration of vessels, causing softening of the anterior horns and intermediate gray matter. This process was accompanied with secondary congestion, dilatation of small vessels and capillaries, but no hemorrhages. The condition was one of softening of the cord precisely analogous to the chronic softening of the brain.

It is not inflammatory, and cannot be called an anterior poliomyelitis. It is not a primary cell-atrophy, and does not, of course, belong to the spinal forms of progressive muscular atrophy.

While the change was, no doubt, largely a senile one, the cord does not correspond to the descriptions of senile cords given by Leyden. Undoubtedly, the feeble gait, loss of knee-jerk, weak bladder, and constipation of senility are dependent upon less marked form of spinal-cord softening than that described here. The disease may be called a progressive senile paraplegia from softening of the anterior horns, due to obliterating arteritis.

Such is my interpretation of the above case; and I have made it while bearing in mind the possible view that the cell-atrophy is primary and the vascular changes secondary.

INFLUENCE OF THE NERVOUS SYSTEM ON PUTREFACTION.

Ibid. Brown-Sequard sent the following note to the Société de biologie: The head of a guinea-pig was crushed instantly with a hammer and the brain reduced to a jelly. The action of the spinal cord was thus vigorously influenced. Rigor mortis was delayed, and putrefaction did not take place in the intestine till the fourth or fifth day.

TWO CASES OF SYRINGOMYELIA.¹

By JOHN AMORY JEFFRIES, M. D.

FEMALE, single, twenty-five years old; sick for ten years; lateral curvature; paronychia; muscular atrophy of right arm, much diminished senses of heat, cold and pain in right arm, neck and side of thorax; spot of same on left thigh; touch perfect; left pupil larger than right.

November 14th, 1889. M. C., with no family history of nervous disease; by nature left-handed; began nine or ten years ago to suffer on sneezing, from a very severe stabbing pain, radiating over the body from about the second dorsal vertebrae. Six months later it became difficult to hold a pen in the right hand, and shortly a "run around" appeared on the thumb. This "was sore, but did not hurt much." After recovery from the paronychia the thumb was wasted; it is not known if the atrophy has progressed. Cuts of the right hand have healed slowly, and corns and blisters appeared from time to time. During the last six weeks has suffered from headache on and off; otherwise has felt well, and has been steadily employed as a waitress in a restaurant.

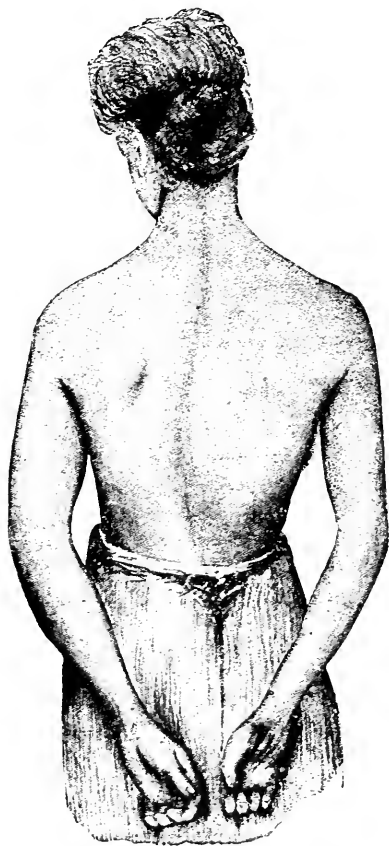
Status præsens.—A slim woman of small stature, expression bright, intelligent, but old for her age. Face symmetrical except for a possible flattening of the right cheek, teeth lost on this side. Motions of facial muscles both from volition and emotions symmetrical. Motions of eyes normal. Left pupil a trace larger than right under all conditions. Pupils react to light, accommodation, and irritation of skin of neck. Tongue projected straight, steady, no fibrillary twitching, of healthy appearance.

Lateral curvature of spine to the right in neck and upper dorsal region; right breast hangs considerably below the left, otherwise the thorax appears normal. Physical examination of heart and lungs showed nothing abnormal.

Right arm smaller than left, which careful examination shows to be perfectly normal. Complete atrophy of the

¹ Presented to the American Neurological Association Annual Meeting in Philadelphia, June, 1890.

flexor muscles of the ulnar side of the right forearm, the periform bone is freely movable. Left forearm normal. Right hand in position of main *en griffe* with complete atrophy of all the muscles except one head of the flexor brevis pollicis and a few fibres in the adductor pollicis; two corns on the palm. In spite of the extensive paralysis, the fin-



From a photograph of M. C., showing spinal curvature and atrophy of right arm.

gers are quite strongly moved and the hand efficient, as shown by her occupation. Left hand normal except for a curious hollow in the ulnar border just proximal to the base of the little finger. I do not regard this as pathological. Abduction of digit is strong and the same appearance is

occasionally to be found in healthy people. Fibrillary twitching of all the muscles of the right arm, none anywhere else.

MEASUREMENTS IN CENTIMETRES.

	L.	R.
Arms at axilla, - - - -	22.2	20.3
" " middle, - - - -	21.6	19.7
" " above elbow, - - - -	19.7	18.4
Forearm, - - - - -	20.0	15.8
Wrist, - - - - -	13.3	12.0
Knuckles, - - - - -	17.1	16.5

Reaction to faradism tested with a Barrett battery, two cells; the figures being the number of millimetres which the fine secondary coil overlapped the primary. Stintsing's test electrode. Erb's largest on back of neck.

Supinator longus of right arm,	17;	left, 12.
Biceps of right arm,	20;	left, 20.

Reaction to galvanism gave K.S.Z. > A.S.Z. in left arm; and A.S.Z. > K.S.Z. in right arm, as tested with Erb's electrodes the indifferent on back part of neck, and a Hirschmann galvanometer. This galvanometer on switching the current back and forth, the circuit remaining the same, showed no changes in the strength of the current. The test was made by slowly reducing the current and reversing until K.S.Z. or A.S.Z. failed to occur. The muscles in the two arms were compared one by one.

Three weeks later tested with an Edlemann galvanometer and Stintsing test electrode, no degeneration was found, but as follows:

Right supinator longus,	K.S.Z.	1.8 m.a.	A.S.Z.	2.6.
Left " "	K.S.Z.	1.7	A.S.Z.	3.5.
Right biceps,	K.S.Z.	1.1	A.S.Z.	3.5.
Right stena muscles,	K.S.Z.	4.5	A.S.Z.	7.
Left " "	K.S.Z.	3.	A.S.Z.	8.

Also K.S.Z. was found greater than A.S.Z. with the same method and apparatus used in the first test. Therefore, either there was some error in my first test, or the reactions changed. The switching of the poles was too frequent and irregular to allow of a uniform error in reading the poles. The same frequent switching would seem to exclude any regular increase of the positive pole by return current in the right arm.

No increase of mechanical excitability of muscles; no triceps or biceps tendon reflex procured on either side.

Organs of abdomen normal.

Left knee-jerk > right; achilles tendon reflex slight, same on both sides; no ankle-clonus; no atrophy of muscles.

Right arm, quarter of body, side of neck and left leg feel "numb;" shown in figure by stippled area. The sensation seemed to be one of discomfort or consciousness rather than of true numbness.

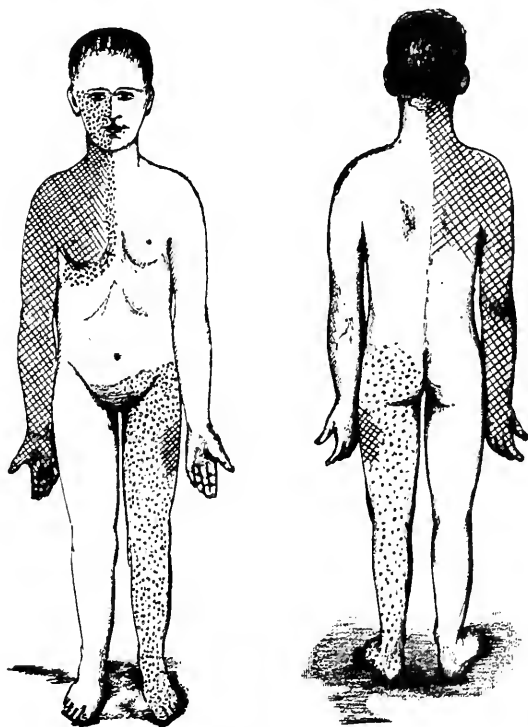


Diagram showing areas of impaired senses of heat, cold and pain (lined), and discomfort (stippled).

Sense of touch perfect all over body, not delayed. Fingers, head and point of pin and wisp of paper used in testing. Sense of temperature, as determined by the use of dry test-tubes filled with hot and cold water, diminished in an area, including the whole right side of the body from the line of the lower jaw to the seventh rib. Line of demarcation from the left side close to the middle line, distinct, but not an actual straight line. All over this area cold

(45° F.) is recognized about 70 per cent. of the trials, the percentage of error being highest an the forearms. No idea can be formed from short contact, some thirty seconds to a minute being required. A correct decision given after half a minute may be reversed at the end of a minute. With heat (130° F.) the percentage of error is higher, nearly 50 per cent. Like cold, the impairment is most on the forearm. No differences between the surfaces. The finger-tips are less affected than the rest of the limb. Patient says there is a small patch on the outside of the left thigh. In all other parts of the body replies are instantaneous, always right even with a difference of a few degrees.

The sense of pain is diminished over much the same area as the sense of temperature, but apparently varies more or less from day to day. The diminution is most marked in the right forearm. The patch in the leg is not very marked. Tests were made with the faradic wire brush, deep pinching between the fingers and working skin between the nails. Over the rest of the body there is certainly no analgesia.

Sense of position of parts perfect, as shown by recognition of slight, slow, passive motions of relaxed parts.

Location of all cutaneous irritations perfect. With eyes closed weight of objects held either one in each hand or one after the other, in either hand, very accurately determined. With faradism, painful cramp in muscles at same strength on the two sides. Sensation of mouth normal. Sight, hearing, taste, smell, good. No ataxia, no tremor, no Romberg symptom, no nerve tenderness.

Thanks to the kindness of Dr. S. G. Webber, I am enabled to report a case of syringomyelia, with autopsy, which occurred in the Boston City Hospital in 1882. As will be seen, but very few symptoms referable to the spinal cord existed, though the lesions were extensive.

"November 14th, 1882. K. D., 31 years old, single, dressmaker. Had lung fever when a child. Curvature of spine since 17 years old. Last fall caught cold after slight exposure and had a sore throat. Soon after had pain in the small of the back, shooting into both hips. Pain in back lasted about a month. Since then constant pain about dorsum of right iliac bone. Of late pain along upper border of the right scapula. At first there was pain in the left hip for three weeks. Cannot walk on account of pain above right hip, but thinks legs are as strong as ever. Limbs and

body much emaciated. No cramps or twitching; no peculiar sensations in limbs.

"No cough, dyspnœa or palpitation. Appetite poor; gaseous eructions; frequent vomiting, usually about two hours after eating; no pain in abdomen. Bowels kept regular by medicine. Micturition infrequent. Frequent sweating. T. 98.3; P. 100.

Nov. 15th.—Pain and tenderness on pressure at right sacro-iliac synchondrosis. Less tenderness over upper part of scapula. Much pain on pressure on the left side of abdomen, seems to start the pain on the right more than hurt at point of pressure.

Nov. 22d.—Nose bleed during night and continuous, less this morning.

Nov. 23d.—Nose bleed stopped yesterday morning. Now has tenderness over the sternum, which seems concentrated on the right near the junction of the fourth and fifth ribs. No fluctuation or sweating.

Nov. 29th.—Nose bleed renewed this morning.

Nov. 30th.—Only slight epistaxis about 6 A. M., on coughing.

Dec. 1st.—Slept badly.

Dec. 2d.—No abnormal sound in heart or lungs.

Dec. 7th.—Still complains of great pain in chest.

Dec. 10th.—Considerable pain this morning. Some œdema of feet.

Dec. 11th.—Nausea and vomiting.

Dec. 12th.—Urine acid, fawn-colored, laden with urates. Sp. gr. 1024. No albumen sediment (after dissolving urates), uric acid crystals and normal epithelium.

Dec. 14th.—Has considerable pain in chest.

Dec. 15th.—Left leg numb, cannot move it well, but makes all the motions.

Dec. 16th.—Has some shortness of breath.

Dec. 19th.—Died at 5.30 P. M.

The pathologist's diagnosis ran as follows: "Chronic thickening of dura. Chronic internal hæmorrhagic-pachymeningitis. Anæmia of brain. Apparent dilatation of central canal of cord. Lateral curvature of spine. Chronic parenchymatous myocarditis. Double hydro thorax. Oedema of lungs. Chronic passive congestion of spleen. Tumor of ovary. Cancer of stomach and neighboring lymph glands."

The only parts of the pathologist's full report of interest are: "Marked curvature to right, little or no compensatory curvature in lumbar region." "Cord showed throughout its whole length a central opening varying in size from two to four millimetres." "Bodies of vertebræ occupying the seat of most marked curvature showed a soft, reddish-gray appearance on section."

I add the result of my microscopic examination of the sections lent me by Drs. Webber and Knapp.

The spinal cord was evidently not well hardened, consequently the sections are thick, and in places there is considerable shrinkage about the vessels. The sections are all stained with carmine, except one stained with hæmatoxylin. Unfortunately the sections are not marked as to level, but only by an arbitrary series of numbers from above down, nor is there any way of distinguishing the right from the left side. Therefore my statement of levels is only an inference from the structure of the sections. One side of the cord can be distinguished from the other by the continuity of the lesions, therefore, to facilitate description, one side is *arbitrarily* called right, the other left.

All the sections from the upper cervical to the lower lumbar region show changes in contour. In the upper sections the cord is flat and wide (14 x 8 mm.), in the lower part this is not so marked. The anterior fissure is wide, gaping, and the anterior columns do not bear their proper relations to each other, but are as it were partly unrolled. In the mid-dorsal region, the left half of the cord is misshapen and smaller than the right. From the mid-dorsal down the cord is of nearly normal form.

All the sections from the highest cervical to the mid-dorsal show a cavity, often large, occupying a space between the gray commissure and the posterior white commissure² in the cervical region, the left horn in the dorsal region. There then comes a short interval with no cavity. Lastly, a second cavity begins just to the right and behind

² By this I mean the small bundle of white fibre which run just in front of the columns of Goll from one posterior horn to the other.

the central canal into which it shortly breaks. This cavity is smaller than the first, and fades into the normal or slightly dilated central canal before the lowest part of the lumbar swelling is reached.

In patches throughout the cord the supporting tissue is increased. Of this no mention is made in the detailed description given below.

No. 1.—From cervical enlargement, 15 x 8 mm. shows a cavity 4.17 mm. x .81 mm., bounded in front by the gray commissure, behind by the posterior white commissure, and encroaching at both ends on the gray matter so as to nearly separate the anterior from the posterior horns. Right end of cavity slightly enlarged; left extended as a narrow cleft down middle of posterior horn. Walls of cavity formed by a mass, varying in thickness from .08 to .45 mm., of glia cells. On the outside the cells are closely packed together, have few or no processes and are round. On the inside the cells have small bodies (stained red), and many long, fine processes—that is, have the form of highly developed glia cells. The cells between are in all stages of transition. In places patches of glia cells, some of which have not stained and appear as if softened project into the lumen of the cavity. The same sort of gliomatous growth extends along the fissure in the posterior horn, the processes in most places bridging it over, thus producing a fine lace work.

Central canal filled with a mass of round cells. About forty ganglion cells in each horn; it is clear, however, that most of the group situated at the base, of the horn, have been destroyed.

A small patch in the apices of the columns of Goll, chiefly the left, is infiltrated with glia tissue, stained deep red; a few nerve-sheaths remain, but show no axis-cylinders.

No. 2.—Cervical enlargement 15. x.8. mm. The cavity is reduced to 1.05 x .81 mm, and occupies the central portion of the cord. The walls are of the same nature.

No. 5.—Upper dorsal; 10 by 7 mm. Cavity triangular with apex in left posterior horn, and base well in front, so

as nearly to destroy the gray matter. The left horns are distinctly smaller than the right, as if collapsed, and only contain six motor-cells as opposed to over thirty in the other side. Same changes in the columns of Goll, especially the right, as occurred in the first level.

No. 8.—Mid-dorsal, 10×7 , irregular in shape. Same triangular cavity in left side, but farther back, so as to spare most of the anterior horn. Central canal still occluded.

No. 9.—Lower dorsal, 10×7 mm. The section is torn about the central canal, but shows no cavity.

No. 10.—Just above the lumbar enlargement, 9×8 mm. A long, narrow split ($1.8 \times .13$) in the posterior horn. Walls as in the upper cavity. Central canal open, with a column cell lining.

No. 11.—Apparently same level as last, (9×8 mm.) Cavity in gray commissure breaking into central canal from the right and behind.

No. 13.—Cavity enlarged to $2.34 \times .78$ mm., involving central canal and adjacent parts. All the commissures preserved.

No. 15.—Lumbar enlargement (10×9 mm.). Cavity lozenge-shaped, $.78 \times .52$, at site of central canal, in parts lined with epithelium; apparently nothing more than an enlarged central canal. No gliosis.

No. 16.—Lumbar enlargement, (11×9 mm.) Cavity slightly larger, of same nature. No gliosis.

It is evident that we have here a case of syringomyelia, with a surrounding gliomatous growth. The case, in itself, does not seem to me to show whether the hole is the result of the gliosis, or the reverse, but judging from other cases the first conclusion is the most probable. Indeed, if the hole is not the result of the gliosis, no shadow of a cause is apparent. The cavity is not an enlarged central canal since that structure exists in front. The nature of the lower cavity is not so clear; the lower part shows nothing definite except an enlarged central canal, but in the upper part there is undoubted gliomatous growth and a cavity separate from the canal.

A point of interest is the extensive destruction of the gray matter, especially of one posterior horn, but also in places (upper cervical and mid-dorsal), extensively involving the anterior horns. It is clear that for a considerable distance in the dorsal cord the majority of the ganglion cells were destroyed. The same is true of the cells at the base of the anterior horns in the cervical region. The cavity in the other parts gives one the impression of having expanded and driven the tissue before it rather than to have infiltrated and destroyed the tissue. It is worthy of note that the commissures between the anterior columns, the gray commissure and the narrow band of white substance posterior to the gray commissure are preserved, as well as a connection between the two horns of the same side. This may explain in part the preservation of functions, but how about the lost ganglion-cells?

The degenerated spots in the column of Goll are small, and do not seem to be of so much importance.

The few symptoms which can be referred to the cord seem remarkable when compared with the extensive lesions found, and would imply carelessness on the part of the house officer in writing the records, were it not that many similar cases are on record. Dr. Webber also informs me that the cord disease was suspected, and the patient most carefully examined in all respects, except for senses of heat and cold.

The infrequent micturition and some of the pains may have sprung from the cord, but the other troubles would seem to sufficiently explain them. The numbness of the left leg was probably of central origin. I am inclined to believe that the spinal curvature was of trophic origin. It could not have been the result of cancer of fourteen years duration, nor even tubercular. In either case, a more definite report from the pathologist might fairly be expected.

The first case shows a perfect history of one of the groups of symptoms caused by syringomyelia, and with our present knowledge, must be accepted as such. Since it is only of late years that the disease has received consideration, it may not be out of place to review the subject before passing on to the subject of diagnosis.

Syringomyelia means a hole in the spinal cord extending as a tube up and down for a greater or less extent. This condition very likely results from a number of causes, but seems, as claimed by Schultze,³ chiefly to depend upon the absorption of a more or less dense gliomatous tumor, having a marked tendency to invade only the gray matter and the apices of the posterior columns. The upper part of the cord is also more affected than the lower part. On this peculiarity of location depends our power to diognosticate the disease. The axis-cylinders in the affected area are quickly destroyed, do not remain as in multiple sclerosis.

Somewhat the same sort of cavities, but more extensive, have been found in the cord in cases of sarcoma (Harris⁴). There may also be cases due to myelitis and softening, (*e. g.* Oppenheim,⁵ Silcock,⁶ Van Giesen⁷); cases due to hæmorrhage; cases resulting from imperfect junction of the cord along its posterior border. Cases of enlargement of the central canal, true hydromyelia, do not concern us clinically. I make no positive statement, as the whole subject is still under debate.

Owing to the peculiar localization of the disease, a definite group of symptoms is produced which renders a diagnosis possible in quite a proportion of cases. The onset of symptoms may be gradual, without cause, or may develop suddenly after a severe fall or the like. The cardinal symptoms are muscular atrophy, loss or impairment of senses of temperature and pain, the other senses being normal, and trophic disturbances varying from bullæ to necrosis of phalanges, deep cellulitis and the like. The upper part of the body is chiefly affected. The legs are apt, owing to descending degeneration, to become spastic-paretic. In the arms the atrophy is frequently of the Aran-Duchenne type, destroying all the small muscles of the hand, and then

³ Schultze, *Zeitschrf. f. Klin. Med.*, No. 13, p. 523, 1887; *Virchow's Archiv* vol. 102, p. 435, 1885; *Ibid*, vol. 87, 1882.

⁴ Harris, *Brain*, vol. viii., p. 447, 1885.

⁵ Oppenheim, *Charité Annalen*, xi., p. 409, 1886.

⁶ Silcock, *Trans. Path. Soc.*, London, 1887-88, p. 18.

⁷ Van Giesen, *Journal of Nervous and Mental Disease*, 1889, p. 393.

working up by regions, not by nerves. Main *en griffe* appears, but the hand is much more useful than might be expected. Fibrillary twitching may or may not exist, when atrophy is complete no reaction occurs. Where fibres are left K.S.Z. may be greater or less than A.S.Z. About this time or before, patient rarely knows when, the senses of pain and temperature are impaired or lost. The change may be one of simple diminution only, differences of five or ten degrees being noted; or complete, severe injuries from burns and scalds then resulting. Sometimes the sense for higher temperature is lost, say from 35° C. up, while that for lower temperature is retained fairly well. The analgesia may be extreme, such that necrosed phalanges can be excised without pain. A hand, arm, quarter of the body or more may be affected, the area of loss of pain and temperature senses roughly coinciding.

It is worthy of note that the areas of impaired sensibility follow the distribution of such troubles in hysteria, that is, involve a hand, forearm or arm, not the radial or ulnar border of the arm as in trauma of the cord.

The sense of discomfort in the affected limbs, as described in case I., may be due to the brains missing the sensations of heat, cold and pain, the equivalent of "vision obscure" of the French.

The impaired sensibility leads to the infliction of many injuries and their subsequent neglect, which is probably responsible for many of the "trophic" changes. But there are apt to be œdema, formation of bullæ and the like, of undoubted nervous origin. In most cases more active inflammatory troubles occur, as loss of nails, necrosis of phalanges, deep inflammation in the arms, necrosis of arm bones or similar processes in the feet. Sometimes active inflammatory troubles form the leading symptom, the atrophy is slight, the impaired senses not noted.

Spontaneous painless fractures of the bones of the upper limbs is also a peculiar symptom, at times the only conspicuous one. Patient, usually a powerful man, comes to the hospital with a broken ulna, radius or metacarpal, with a history of having suddenly lost control of the mem-

ber some days before. It is the swelling or uselessness which brings him in, not pain for there is none. Such fractures are apt to do well—knit as in healthy people. In some cases there may be a trophic change in the bones, but in Schultze's case with autopsy, there certainly was not. These fractures seem to be due to the unconscious use of extreme force, a point of some interest, as they occur in cases where senses of touch and position are normal; sense of resistance not noted.

The legs may show precisely the same symptoms, with or without the arms being affected. But owing to descending degeneration from the more advanced changes in the upper part of the cord, are apt to be more or less in a state of spastic paresis. At times the two classes of changes are blended, producing a confusing symptom complex.

A prodromal symptom not rarely noted by the patient and present in many cases, is that of pain in the spine between the shoulders and radiating over the body on sneezing. Neuralgic pains often occur, located chiefly in the affected regions. The pains may be furious, all sorts of paræsthesia are frequently present and may go far toward making life miserable. Such are the symptoms which are of value for diagnosis, many others are liable to occur, almost any which can result either from irritation or destruction of parts of the cord. Thus sometimes early, frequently before the end, the sense of touch is seriously impaired, also the sense of position, resulting in total anæsthesia, or hyperalgesia may exist.

The head is not much affected unless some secondary or complicating trouble, as cerebral glioma supervenes. Through the sympathetic the pupils may be large and sluggish, the lid droop and the eye be sunken. Towards the end true bulbar symptoms may supervene and carry off the patient.

The bladder, rectum, escape till late, if affected at all.

Death results from general wasting, bed-sores and the like, as in other spinal troubles, from bulbar symptoms, or from septic infection resulting from the inflammatory troubles of the affected members.

It must not be forgotten that the above description is only intended for that class of cases which run a typical course, the group which is recognizable. There are a good many cases showing the symptoms of multiple tumor, myelitic fossæ and the like, which are not recognizable; also cases as with cerebral tumor occur without any symptoms. Of this case II. is a marked example.

DIAGNOSIS.

Where the three cardinal symptoms of atrophy, loss of senses of heat, cold and pain exist, the difficulties of diagnosis, barring a few diseases, is slight.

The diseases which appear to need special consideration are pareso-analgesia of Morvan, hysteria, leprosy, neuritis, progressive muscular atrophy and small focal lesions in the posterior part of the internal capsule.

Morvan, from a small district in France with a population of 50,000, has described a group of some seventeen cases which closely resemble cases of syringomyelia. Schultze and others have pronounced them to be cases of syringomyelia; this Morvan has steadily denied. According to Morvan a distinction is to be made by: 1. The much more marked trophic troubles and arthropathies. 2. The constant impairment of the sense of touch.

A study of the history of his cases certainly gives a different impression from that of a similar number of cases of syringomyelia. The muscular atrophy seems to be less pronounced, the legs are comparatively exempt, and the trophic troubles distinctly more pronounced. Not a few of the descriptions might be taken for partial descriptions of leprosy.

While it is too early to make any positive statements in regard to pareso-analgesia, Morvan's position is certainly supported by an autopsy made by Gambault, in which a peculiar thickening of the nerves, a sclerosis of the cortical zone of the cervical cord, and thickening of the arteries

* Morvan, *Gazette hebdom. de Med. et Chir.*, Aug. 30th, Sept. 6th, 1889; *Ibid* No. 32, et seg., 1886.

was found. In another case the nerve of an amputated finger showed the same sort of changes.

To my mind, Morvan's position is also strengthened by the large number of cases occurring in a small population, while syringomyelia is a rare disease of general distribution. It certainly looks as if *Mal de Morvan* might be hereditary or due to some habit, local custom or occupation, but time and autopsies alone can settle the question.

As Charcot⁹ has lately pointed out hysteria can produce a symptom complex closely resembling syringomyelia, as special paralysis of the senses of pain and temperature, paralysis, trophic disturbance and atrophy. One or two of the reported cases of syringomyelia certainly read very much like hysteria. Hysteria should, however, be distinguished by careful observation, history and course. When either disease is at all well marked there can be no doubt.

In my case, as in many others, it might be held that the patient really suffered from two diseases, progressive muscular atrophy and hysteria. But there is none of the hysterical mien or other symptoms of hysteria, while the whole can be explained by one cause.

Anæsthetic leprosy offers considerable difficulties of diagnosis at times, but with care can probably always be excluded. Unfortunately, a clear conception of leprosy is rendered difficult by the inclusion of a certain number of cases of syringomyelia, as the cases of Langham¹⁰ and Steudener.¹¹ According to Leloir,¹² upon whose work I have chiefly drawn, the anæsthesia of leprosy has two origins. In one class of cases it is due to the injury of the terminal nerves in the skin by infiltration, the same as in lupus and other skin diseases. In these cases there is or has been a visible cutaneous lesion, and the affected areas occur in patches. The anæsthesia may be total, be general, but nowhere complete, or affect only sense of pain or temperature. These cases should offer no difficulty of diagnosis,

⁹ Charcot, Bull. Med., Paris, iii., 787, 1889.

¹⁰ Langham, Virch. Archiv., vol. lxiv., p. 475, 1875.

¹¹ Steudener, Beiträge zur Pathologie der Lepra mutilans, Erlangen, 1867.

¹² Leloir, Praise pratique et theorique de la Lepre, Paris, 1886.

especially as a piece of skin can be punched out and examined for the lepra-bacillus, which is usually abundant in the skin lesions. In the other class of cases, the pure lepra nervorum, there is muscular atrophy and partial sensory paralysis due to a leprous thickening in the nerves. At times, touch is normal, senses of pain and temperature being the same as in syringomyelia. Judging by Leloir, the mass of these cases show at one time or another skin lesions which are sufficient to separate them from syringomyelia, but all acknowledge that at times there may be no surface manifestations. None of the authors known to me, however, give clear histories of such cases. Neither the case of Jacobi or Morrow can be so classed.

Granted that such a case, or one in which the skin symptoms have passed off and are denied, there still remain several points of value.

The atrophy and partial sensory disturbance is limited to certain nerves, not segments. Even when all the nerves, say of the hand, are affected, the border is irregular, and judging by descriptions, the sensory paralysis fades away. In leprosy the nerves are thickened, in syringomyelia this is not the case, unless deep inflammation has existed. In syringomyelia, on the other hand, there are apt to be a host of other symptoms pointing to central trouble, as signs of secondary degeneration up or down.

Though Jacobi¹³ enlarges upon the great difficulty of diagnosis he arrives at one which it would be difficult to question. With Morrow's¹⁴ case the conditions are different it would seem, judging by the skin eruption, contractures, loss of all senses, irregular border, to be a case of leprosy, certainly these characters are not special signs of syringomyelia. Unfortunately the description of the case is more or less contradictory with itself and the figure, and also leaves important points in doubt. Progressive muscular atrophy is excluded by the absence of loss of the surface senses.

¹³ Jacobi. *Jour. of Nervous and Mental Diseases*, No. 6, 1886.

¹⁴ Morrow. *Jour. of Cutaneous and Genito-Urinary Diseases*, vol. viii., p. 1, 1890.

Multiple neuritis is said at times to produce more or less similar conditions to those of syringomyelia, but in the great majority of cases there is no partial sensory paralysis. In all cases the disturbance is by nerve areas. Picked sensory paralysis, due to diseases of the nerves, are rare, and but few cases of loss of temperature sense or analgesia are on record in which the pathology is known. The cases of Jacobi and Ziehl¹⁵ depended upon local injury and infiltration of the nerve. Pick's¹⁶ case depended upon exposure to cold, and is by no means clear as to the seat of trouble. The report of Berger's¹⁷ case gives no evidence as to pathology, and by no means excludes hysteria.

Cerebral trouble may produce partial sensory paralysis as well as muscular paralysis. In these cases there is no such atrophy as in syringomyelia, and the paralysis is of the monophlegic or hemiphlegic type.

It is hardly necessary to say that a large number of cases of syringomyelia vary so much from the type as to acquire a special line of reasoning for their diagnosis, provided they do not baffle all efforts.

To those who desire to study up the literature the summary of 116 cases by Baumler,¹⁸ and a recent review by Buhl,¹⁹ will give the requisite start. The American articles are by Starr,²⁰ Upson,²¹ Van Giesen and Booth.²²

¹⁵ Ziehl. Deut. med. Woch., No. 17, p. 335, 1889.

¹⁶ Pick. Wien. med. Woch., p. 617, 1889.

¹⁷ Berger. Wien. med. Woch., p. 786, 1872.

¹⁸ Baumler. Deut. Arch. f. klin. med., xl., p. 443, 1886.

¹⁹ Buhl. Archiv f. Gen. de Med., July, 1889.

²⁰ Starr. Am. Jour. Med. Soc., p. 457, 1888.

²¹ Upson. New York Med. Jour., p. 238, 1889.

²² Booth. Medical Record, p. 236, 1888.

CASES OF HYSTERIA TREATED BY HYPNOTISM.¹

By S. G. WEBBER, M. D.,
Superintendent Adam's Nervine Asylum, Boston.

I WISH to report two cases, which are of interest as being rare. The first is rather a curiosity ; the patient fell into a hypnotic state, seemingly on account of the noises in the house, a state for which I know no better name than autohypnotism, though she did not consciously put herself into that state.

Mrs. S. was born of rather neurotic parents ; her mother had sick headaches, was rather nervous ; her father had bad turns, which as described were probably epileptic. It is not certain that he had these before her birth. There were three brothers and two sisters, the oldest sister has sick headaches and is nervous, the other sister is young, not very well. The brothers are all well.

Mrs. S. never knew what it was to be tired and considered herself perfectly well when she was young. She helped to take care of her father, did much of the house-work, hurried her breakfast so as to get to school in season, studied hard because she liked to ; then she hurried home to learn her lessons and do what she could at the house-work.

One morning on waking she found that her eyes had given out, and she could not open them. When her eyes were better she returned to school, and six months later graduated. During this time she had heavy feeling in her head in the morning, and had the blues. When she graduated she had lost within a few months forty pounds, running down from one hundred and thirty to ninety pounds. She rested a few days, then visited friends with the intention of regaining her health. For ten days she slept much and wanted to sleep more, but injudicious friends thought it would not be well for her. She eat little, her head felt strangely, it seemed to her as though she had been away

¹ Read at the Annual Meeting of the American Neurological Association in Philadelphia, June, 1890.

but she could tell where she was. Noise troubled her, she had nausea, no energy. It was at this time that she had the first spell of unconsciousness. I could get no satisfactory account of the nature of this attack.

She returned from her visit and remained at home for six months, having back-ache, head too tired to sleep, ached all over, lounged about the house. In this condition she took a school, taught seventy scholars for six years. While teaching she did part of the house-work at home, she got breakfast for the family. She had dyspepsia and was depressed in spirits.

While teaching she had the attacks of unconsciousness after unusual excitement, not specially when she was tired from teaching. They lasted five or ten minutes. She seemed to have fainted, but would keep up a rambling talk about what had disturbed her.

She was married about three years ago. After her marriage she did not have many attacks, they came only after great mental excitement. She had one when she learned that she was pregnant. She did not always talk in them. Her husband said that she did not have them when alone.

During her pregnancy she slept a great deal. After the birth of the child she was not able to sleep as well. Her head is weak and gives out easily, she thinks her body is strong.

She did not come to the Asylum until more than two months after the above account was given me. In the mean time her child had been ill and she had had the grippe, so that she had had more to wear upon her and run her down. When she came she was evidently not in so good a condition as when I first saw her, she was more nervous and weaker.

The day after her entrance she had an ill turn after walking into the sitting-room. The nature of this was not clear, it was described as different from a faint, yet she lost consciousness. When I saw her she had partially recovered and was able to talk, but was still dazed, did not understand where she was, said a few words without evident meaning, but which probably had reference to the impressions she had while in the attack, as when a person continues and finishes a sentence after waking from a dream. She had another of these attacks after getting to bed.

On the second day I found that these attacks were of a somnambulic nature; in the attack she did not recognize her attendants, did not know where she was, she did not remember what had taken place only a few minutes before when she was in her normal condition. On coming out of

one of these attacks she did not remember what she had said or done during it ; but when she had another she remembered what had taken place in the previous one, though this memory was not as distinct as the normal waking memory. For a while there was the double existence of which we read in books on metaphysics.

During the following days these attacks of somnambulism occurred several times. While she was in one of these I stroked her forehead, whereupon she went asleep immediately and after a while awoke in her normal condition. The next time she had an attack she called for the hand which had touched her head ; she said that it had a very soothing effect, and she asked every one who came into her room if he had done it. When I entered she had no recollection that I was the one who had soothed her, but when I touched her head she at once said, " That is the hand," and went to sleep. In the normal condition she had head-ache, in the abnormal her head was all right, did not ache. After this I put her to sleep every night and several times in the morning. Only a few strokes were needed to put her to sleep, except when she was more than usually excited : on two or three such occasions it was not possible to succeed.

The manner in which she went to sleep was peculiar. She settled herself down with a contented air, shut her eyes and seemed to be asleep ; then there was a peculiar laugh, and after that she was sound asleep. Once, when much disturbed, she could not get asleep. She then talked, said she could not get way down, that some one made her come up ; she then laughed and seemed asleep, but soon after said again that she could not get way down. This was repeated two or three times. She sank down in bed and stretched herself up in accordance with her words, and she seemed to be much troubled because there was this hindrance.

After a few days she had no more of the somnambulatory attacks but seemed to be in the normal condition all the time, yet it was not perfectly natural, for she could not see me clearly ; she said there seemed to be a mist between her and me.

Two or three times she awoke saying that she could not see ; the first time this happened a few strokes across the forehead sufficed to restore the sight. The other times it was necessary to rub her eyes, and this seemed to cause pain till the sight was restored, after which the same rubbing gave rise to no pain. On one occasion only one eye was blind ; then rubbing the sound eye was not painful, but rubbing the blind eye was. While she could not see, the pupils

were widely dilated and light had no effect upon them ; when sight was restored the pupils contracted normally.

On Sunday, the nineteenth, she saw her husband, who told her of the sickness of her housekeeper. This disturbed her much, and she had a poor night. The next morning I let her tell me all about her trouble and she soon after went to sleep , as usual, after her head was smoothed.

She was disturbed by noises, even comparatively slight noises disturbed her. She was especially annoyed by being waked up about seven o'clock by the necessary noises of the morning. She said it was agony to be thus waked up instead of waking up herself.

On the twenty-first she saw her mother about the housekeeper, and after that seemed easier on that subject. She had two or three good days and nights, sleeping all night and parts of the day.

I tried suggestion in regard to eating. After she had gone to sleep I told her on awaking to be hungry and eat well. She had eaten poorly before. The first time I did this she was hungry in the morning and eat the whole of what was taken to her for breakfast. Several times she did as well and at no time did poorly, except when on account of disturbance she did not sleep well. I also told her to sleep all night. She said that when she half waked up she seemed to hear a voice saying, "Sleep all night," and she did not dare to wake up. She thought this very singular. She may have thought that I had something to do with the voice and spoke of it to find out if it were so. Once when in the daytime I told her to sleep two hours she was awaked by a noise and heard, just as she awoke, a voice saying, "Sleep—hours." She said that in the morning, when the noises of the day commenced, there was a struggle between the desire to sleep and the need of awaking on account of the noise, and this struggle was painful. While in the hypnotic sleep, if there was a noise in the hall, her forehead contracted, and if the noise was loud she shrank and drew up her shoulders as if hurt. If I made a slight noise in moving about she took no notice of it, even though it was as loud as outside noises which she noticed.

She complained so much of the noise that I thought it best to tell her that it could not be entirely prevented ; that we could not keep the house as still as a private house can be kept, and that she sometimes made noises and had waked up a patient. This did not at the time disturb her very much. That night, however, she was not herself (the 24th). She had been all day in an unnatural state, but it was not

reported to me till night. When I went in as usual to put her to sleep, she took me for her husband, called me by his name and began to tell me how she had disturbed the patients; said that he must talk in a whisper, and if she made a noise she would be sent away; that she had done an awful thing in waking up a patient; that she had told him before coming that she knew she would make a noise and would be sent away, and why had she come. She didn't want Dr. Webber to know how she felt, but when he told her that she had disturbed others it went through her like an arrow.

When I tried to put her to sleep she said, "You know it is no use your trying to get me to sleep; you know you have tried to do that a good many times and not been able to. I shall never get well now, for Dr. Webber won't come to see me again, and he won't be able to put me asleep again." Such was the way she talked. I bid her good night and left her. Soon after I heard a noise in her room and went in; she was sitting up in a chair. She still called me by her husband's name. I helped her into bed, told her it was after nine o'clock and I could stay no longer. I obtained a promise from her to stay in bed all night.

The next morning (the 25th) she was still not herself. She told part of the events of the night before correctly and mixed up with these her delusions that she had been carried to another place. I was no longer her husband, nor was I Dr. Webber.

It is unnecessary to detail what she said. There was about the same coherence that is found in a well-connected dream. In all the time that she took me for her husband she did not treat me with any undue familiarity; she was as ladylike in her manner of speaking and acting as if I had been a stranger. There was no surprise nor sense of inconsistency that I did not show any more interest than a stranger, nor meet her as warmly as a husband might. Either she was not sure who I was, or, as in a dream she did not notice any incongruity.

In a few other cases I have had entirely different experiences. The patients have not been satisfied without some manifestation of affection.

After allowing her to tell me all she seemed to wish on the morning of the 25th I kept my hand on her head. She took no notice of my smoothing her forehead, trying to get her to sleep, so I simply kept my hand quietly on her head. At length she suddenly put her hand on mine exclaiming, "What's that!" then she began to sob, turned

over and went to sleep. In about an hour and a half she woke in her normal condition. She remained herself all day and at night professed to have no recollection of having seen me in the morning. She seemed to have lost all memory of what had happened the previous night. She went to sleep readily.

During the day of the 30th she was moved into another room in hopes that the noise would trouble her less. That night she did not sleep well as usual, and the next day had a severe headache all day. When I saw her at night I held my hand on her head a few minutes and she said the pain had gone ; she was free from it.

That night she slept only two or three hours, and the next night it took a long time to get her quiet, and she slept only half an hour. She seemed worse, the noises disturbed her so much that I advised her being taken home. She was not gaining, but rather getting worse. Subsequently she gained in weight and was able to sleep. As she grew stronger noises troubled her less.

Miss F., aged twen y, says that she has had dizzy headaches very often ever since she can remember. She rarely went a week without one. She thinks she was well as a baby.

Her mother was nervous, had neuralgia ; she has four brothers and sisters ; they are all well except her youngest brother, who has rickets. Her father is intemperate, so she cannot live at home.

She had measles when young and scarlet fever at eight or nine years of age ; no other illness. Using her eyes made her headache. She left school at seventeen in poor health on account of headaches. Otherwise she had good health. After leaving school her condition remained about the same ; she would have headache two or three times a day for a while, then would go several days without pain. The pain was across the forehead and in the temples ; there was no nausea ; sight was blurred ; she was dizzy.

Last June (1889) she fell from a swing, striking the left side of her head ; she had a faint feeling and dropped out of the swing ; she does not know what distance she fell. For a minute she was unconscious. After this her head ached for a week and felt tight on top, very sore on the left side. The left cheek was discolored, and this extended down the left side of the neck ; her back troubled her for a few days after. After a week the headache would go and come. Her work was not hard ; she had a large appetite ; slept well.

From Thursday to Saturday she had a dreadful headache all the time.

On Sunday, the nineteenth of September, she felt better and went to church. She had been in church but a short time when she became dizzy ; things moved, dancing about ; she felt herself falling and couldn't save herself. She remembered nothing after this till she came too in the vestry ; then she felt tired, her head seemed tightened across the top. She was taken to her room and put to bed ; that afternoon she had another in which she was violent, straightened out, threw her arms about, not her legs ; she threw herself backwards and forwards very quickly, then she stiffened out straight. Her eyes were sometimes open, sometimes shut ; at times they were crossed, always "set." This attack lasted an hour. After the second her head ached dreadfully, all on top, and it was very sore there.

She went to work on Tuesday, and in five or ten minutes she had the same feeling of things growing black, and felt she was losing herself. She had an attack in the office. After this she was sick in bed two and a half weeks ; she had attacks once a day, or even twice.

The Saturday after the first attack she had three severe attacks, one lasting three hours. She first worked her muscles all over for ten or fifteen minutes, then was rigid and then lay passive, the face and hands moving a little, the fingers closed and the fist rotating.

The following week she had one every two or three days, lasting an hour and a half. During this time there was considerable mental excitement apart from the illness, which must have acted to increase her nervousness, and probably caused the attacks to be more frequent. She went into the country ; had one on the train ; the next day one, and after that only at the catamenia, except when she had the grippe at Christmas. That night she had one lasting three hours, the next day one of four hour hours duration, and in the afternoon one lasting two hours. Two the next day.

On the sixth or seventh of February she had one at the catamenia as usual. She was taken to the New England Hospital and had five during the week she stayed there. The menstrual flow continued the whole of that week, but usually it lasts only two or three days, and generally she has only one at each period.

Between her periods she has severe headache, not every day, and after an attack the pain is behind her eyes, under the brows, and shoots to the top of the head. There is no

increase of micturition after the attacks. Just before a headache her face is flushed, and it seems as if all the blood rushed to her head.

Her appetite is poor and she does not sleep well. Bowels are constipated; she has no leucorrhœa. She was examined by a female physician, who found no uterine disease.

On March 9th she had three attacks. This was the time for her catamenia, but there was no flow. During the next week she had two severe headaches on the top of her head. Phenacetine powders relieved the head each time, and afterwards it felt light as if the top were gone. After the 9th she felt ill for three days, but did not flow.

I saw her after one of the above attacks. She was then much weakened, and had the appearance of being exhausted. The attack was described as one of the worst she had ever had. She was delirious most of the night; it required three or four women to hold her and prevent her leaving her room. During the convulsive stage she was more violent than usual.

About a week after these attacks I began to hypnotize her. The first trial had no effect. She simply became quiet. After the third time she went to sleep in my office, but it was a troubled sleep. After a few treatments she slept quietly without dreaming, as in natural sleep. Most of the time during her sleep at my office there was more or less twitching of the muscles of the chin and face, especially on the right side. She said that while going to sleep she felt herself sinking down gently, and just as she lost consciousness she felt that she had reached the bottom. This sensation was not unpleasant, but rather agreeable, and seemed to resemble that of which Mrs. S. spoke. When she is asleep I can make any noise about the room without disturbing her, even throw a book on the uncarpeted floor with considerable force without awaking her, but at first if I spoke she woke up. After I told her not to mind my voice she was less easily disturbed by it. A touch would arouse her at any time very quickly.

During this time she was taking sixty grains of bromide of potassium a day. Notwithstanding this she did not sleep well the nights when she had not been hypnotized, but the nights after a treatment she slept well, often eight or nine hours. Her room-mate said that she slept much more soundly than before the treatment was commenced; formerly she would jump in her sleep, her limbs were rarely quiet, and she was much disturbed not only by anxiety lest

there should be a spasm, but also by her restlessness. After the treatment had continued a week or ten days this restlessness disappeared and the sleep was quiet. The patient's countenance changed for the better ; the eyes lost a stupid look they had, the nutrition became much better and there was a general appearance of health which was not present at the first. The patient herself said that she was stronger, could walk longer distances without fatigue, and felt more like working.

April 19th I hypnotized her and allowed her to sleep longer than usual. I thought I would see how long she would sleep if not disturbed. It was just the time for the catamenia. After fifteen minutes she woke, seemed more confused than usual, said that her head ached, then went into a spasm. The convulsion began with a twitching of the chin and right side of the face, next there was a stiffening of the body and rigidity of the arms with tremor, the hands being clenched. There was loss of consciousness from the first sign of an attack. In a minute or so there was spasm of the muscles of the back causing opisthotonos, the hands were extended and in extreme pronation with the wrists flexed. Pressure over the ovaries seemed to cause relaxation at once of the spasm, and she lay relaxed and limp. The right side of the face and the right hand still twitched. Her eyes opened for a few minutes, but she did not seem to see. After about twenty minutes she had another attack much like the previous but less severe, lasting only about three minutes. After this second attack the face twitched and the hands were slightly convulsed, mostly on the right side ; the legs were quiet. In forty-five minutes from the beginning of the attack she was conscious. Her friend said it was a very light attack.

In four days she was able to come to my office again. The catamenia had not appeared. In ten days that function was established and she had a very light attack of only a half-hour's duration, and so light that she did not know she had had one. She slept for two hours after it. She thought that the attack was due to having her sleep disturbed by a toothache, and that caused a headache.

When she came to me the next time she had been to the dentist and had had the tooth filled. It still ached much with a jumping pain. When she went to sleep I suggested that on waking the pain should be gone. On waking she had no pain, and it returned only once for a few minutes. On other occasions I suggested that pain in one part or another of the body should leave her, and on waking it was gone.

About a week before the time for her last catamenia I suggested that she should come round at the regular time (she had been two weeks late the two previous periods) and that she should have no convulsions. I made this suggestion each time I hypnotized her. She was only two days late and had no attack, and scarcely any headache. At the next menstruation there was no spasm, it was normal and nearly at the right time. The bromide had been reduced to a small amount taken only a week before the catamenia.

The first is a complicated case. There was a peculiar condition of the nervous system, a condition of neurasthenia, also a hysterical state. In some way, which I do not understand, the patient came into a state in which she lost her memory of recent events, and her relation to those about her were changed to such an extent that she did not recognize those whom she had seen and known only a few hours previously. Then she returned to her normal condition and had no recollection of what had just occurred. Her brain then became a dual organ; in some way the awaking of one set of ideas and associations in one group of nerve cells seemed to inhibit the activity of the group of cells which was cognizant of the other set of ideas and associations.

Later when I had put her to sleep there was the manifestation of other phenomena; there was not only the two states of brain activity, but also the power or ability of receiving suggestions when all the mental faculties seemed to be dormant, and it would have been supposed that she could not hear nor the brain receive any impression, at least so as to have a permanent effect.

Memory, consciousness and will were perverted, or in the artificial sleep they seemed to be abolished; certain groups of nerve cells, which are usually active during waking hours, were inhibited in their action. For a while they were placed in an enforced quiet. This was seen in the second case. I could easily put the patient to sleep; there was no cataleptic state of the muscles; she simply went to sleep, a quiet sleep such as any one might have, with dreams or without as the case might be. She was, however insensi-

ble to all noises, was not disturbed by the rattling of paper, opening of table drawers, the slamming of doors outside, nor by my moving about the room. As soon, however, as I spoke to her, she showed that my voice reached her brain, there was a change in the expression of her face, and if I continued to speak she soon waked up.

It will be interesting to compare these peculiar mental conditions with what is seen in an ordinary case of delirium, in which there is a more or less complete unconsciouness of one's surroundings. I will report a very simple case which resembles hypnotism.

Miss A. has had severe headaches, especially at the time of her catamenia, and in these is sometimes delirious.

March 20 was called to her about seven in the evening, as she was delirious. I found her lying on her right side ; the muscles of her neck and arms would occasionally twitch and she carried her hand to her neck every now and then, and showed by slight groans and sighs that she had severe pain. She seemed to imagine herself at home, and called her mother in a rather low tone of voice. When the nurse came she told her to go for her mother. She seemed to think I was her uncle Edward. She tried to get up once or twice, and said she must go or be going. There was no violence and no loud outcry ; she was simply in a quiet delirium. She did not realize where she was nor what she said, yet seemed to realize that she was in bed, and tried not to uncover herself. Under the use of remedies and massage to the head she fell asleep, awoke at a slight noise, then went to sleep again, and soon woke up in her right mind, astonished to find me by her, and asked if she had done anything out of the way.

In this case there was an entire misapprehension of her condition and surroundings. She was as much out of touch with those about her as the hypnotic, more so in fact. Yet she retained a certain amount of receptivity ; she knew that someone was near, and made known her request that her mother should come. In this patient the memory and consciousness were perverted or suspended, the will was still active, but acted in an abnormal way, because it had not correct data to guide it. Ideation was more affected. In hypnotism there was a more orderly sequence of ideas ; in

delerium the sequence of ideas was less regular ; slighter influences served to give rise to new ideas not in harmony with those preceding. It seems to me this may be one of the differences between hypnotism and delerium ; not only are memory, consciousness and will affected, ideation is irregular and dependent upon accidental influences, arising outside or perhaps within the patient. In hypnotism, when ideation is affected, it is perverted according to the will or suggestion of another.

It would be interesting to compare these two states with dreaming. This seems to be a modification of the above conditions, the cause being within the brain itself, or often coming from without. But in dreaming there is less order, the control of the thoughts, which is not wholly lost in hypnotism, and which still is found to some extent in delerium, is very imperfect in dreaming ; the logical faculty is more affected, or it may be said the memory is more at fault and does not act to regulate the sequence of ideas, so these run riot in the brain, and the different groups of nerve-cells are awakened to activity and silenced in a most unordered manner, giving rise to the most absurd and incongruous grouping of ideas and images.

Dreaming is likely then to be the least orderly of these abnormal activities. There are to be sure dreams, which seem to be perfectly orderly and logical. Perhaps they are so, or it may be that they seem to have this order because the waking mind unconsciously reduces the mass of ideas to something approaching order. It would be interesting to follow the analogies in these states much further.

A CASE OF FOCAL HEMORRHAGIC LESION OF THE PREPEDUNCLE (ANTERIOR CEREBELLAR PEDUNCLE.)¹

By H. M. BANNISTER, M. D.

Kankakee, Ill.

HEMORRHAGES, or other lesions of the anterior cerebellar peduncles appear to be among the very rarest of pathological accidents. The following case is therefore considered as worthy of being reported.

R. W., American ; aged 37 ; married, and mother of one child ; was admitted to the Illinois Eastern Hospital for the Insane, Oct. 1, 1889, with a very meagre history. All that could be learned was that her parents were first cousins, that she had a rather worthless husband, had been always well prior to her confinement, that she had been insane for several years, cause unknown, and that for some time she had not changed in her mental or bodily condition. On admission she was in good physical condition, well nourished, no paralysis or motor disorder whatever, special senses and their organs apparently all normal, menstruation regular. The only abnormalities were a slight papular eruption (stated as "acne papulosa" by the examining physician) and a systolic cardiac murmur.

When she came under my care shortly after, there had been no change ; she was a typical untidy terminal dement, stupid and filthy in habits, even with close personal attention, but not violent or hard to control. She seemed to understand simple orders but seldom spoke, and then only to herself, and incoherently. There were no special troubles or

¹ Presented to the American Neurological Association Annual Meeting in Philadelphia, June, 1890.

peculiarities of speech and no decided indications of organic brain disease. Her appetite was generally somewhat excessive, and her general health very good.

On the morning of March 7, 1890, she rose as usual and ate a hearty breakfast and appeared quite in her usual health till 8.30 A.M., when she was suddenly taken with an attack of vomiting, something entirely unusual with her, and had at the same time a passage from the bowels, soiling her clothes, which was less out of the usual course of things, though in this instance it was probably involuntary. When she was taken to the bath-room, the attendants noticed that she walked with difficulty, and as they described it, "she acted as if she was dizzy." The vomiting was rather persistent, and she was put to bed and word sent shortly after to the medical office, though they did not report the case as at all urgent. She had to be helped by two attendants and they reported that in going upstairs to the dormitory, aside from her reeling she leaned rather heavily to the left.

Owing to other calls I did not see her till nearly eleven o'clock. She was then lying on her right side, her face was slightly flushed, eyes open, and pupils about normal, neither specially dilated or contracted, pulse a little accelerated, 80 or more, tongue lightly coated, temperature normal. She was conscious, could move herself in bed, and gave no signs of being in much pain, but had vomited freely since she was put to bed. There was nothing in her appearance or symptoms as she laid in bed to make me feel sure there was anything more the matter with her than some gastric irritative disorder, so I left a prescription suited to such, and gave instructions to have her watched and any change reported.

About the middle of the afternoon word was sent that the patient was worse, and I saw her again at 4 P. M. She was then lying with head strongly turned to the right and her body partially turned in that direction, a semi-lateral decubitus; her face was flushed, some frothy matter about the mouth, as if she had been vomiting or had had a convulsion (but the attendant had observed none), her eyes tightly closed, respiration rather rapid, 25 to the minute, and accompanied with mucous rattling in the throat and

coarse rales in the bronchial tubes. It occasionally slackened in its rate for a few seconds and then became rapid again, but this was not entirely regular. The temperature taken in the axilla was not above normal. Pulse fairly full and strong, ranging from 90 to 100. When the eyelids were forced apart the eyeballs were seen to be in rapid horizontal movement to and fro; the pupils were moderately contracted, their apparent diameter was about three or four millimetres. The left arm was rigidly flexed over the chest, the left leg was extended and lax, and though well protected by the bedding was decidedly cold to the touch, while the other parts of the body were warm. Sensation was apparently entirely lost in the left leg, and the plantar reflex could not be excited, while it was very marked in the right. There was no sign of facial paralysis. The appearance of the patient, aside from the tight closure of the eyes, which seemed a voluntary act, was that of complete coma.

The attendant stated that her condition had been gradually changing since dinner time (12.30 P. M.), when she had tried to eat but could keep nothing on her stomach. She had also soiled herself once since my former visit and her kidneys had acted freely.

From this time on the patient's condition grew progressively worse, the whole left side became paralyzed, the breathing stertorous and puffing, the decubitus dorsal, the pulse weaker and more rapid, the right side also parietic, and the urine and fæces were passed involuntarily. At 10 P. M. the head was still turned to the right, and the eyelids were closed, but not tightly, and the nystagmus was not observed. The left side, especially the leg, was palpably colder than the right, and this difference was very noticeable till the last hour or two of life, when it became somewhat less marked. The night watch reported that there seemed to be some fever before midnight, but the temperature was not taken. During the latter part of the night the patient lay quietly on her back, the limbs and muscles generally in a state of complete relaxation, and after 1 A. M. the respiration changed in character, became irregular, at times

rapid, and then almost intermitting altogether. She grew steadily weaker and died very quietly at 7 A. M. on the 8th.

Owing to failure to obtain the consent of friends, the autopsy was delayed till twenty-seven hours after death when it was finally concluded to make at least an examination of the brain. This was made by Dr. H. D. Valin and the writer, on the forenoon of March 9th, with the following results :

Body well nourished ; considerably discolored by sugillations. Nothing abnormal in the external integument of the cranium. On removal of the skull-cap the skull was found to be of about normal thickness, but the inner table was abnormally thin and porous looking. The dura was free over the whole convexity of the hemispheres, except at one or two points near the falx, and seemed dark in color and thinner than usual. Its vessels were also unduly noticeable and tortuous.

The vessels of the pia were enlarged and engorged, the arachnoid thickened and opaque in places. This was especially the case on the right hemisphere in the vicinity of the Sylvian fissure and over the superfrontal and part of the medifrontal gyri, from the apex of the frontal lobe to the central fissure. At the upper end of the pre-central gyrus, about a centimetre from the median fissure there was a nearly circular adhesion of ten or twelve millimetres in diameter in which the cortex tore away with the separation of the dura. The whole brain was in a very softened condition, so that it tore very badly in handling, and especially in its removal from the cranium, in spite of care, but aside from the post mortem injuries thus received there was nothing noticeably abnormal on the basilar surface of the brain.

No gross lesions were found in the hemispheres or basal ganglia, except a few dilated vessels and a small vacuole, about the size of a millet seed on the margin of the lenticula, and the anterior limb of the internal capsule. This was observed on one horizontal section about the level of the aula. The ventricles were rather pale, and I could not say *largely* distended. The pons, medulla and crura, though

badly softened and with some of their minuter gross anatomy obscured from this cause, showed no macroscopic lesions, and the same was the case with the cerebellum, which also appeared to be less softened than the other intracranial organs.

On the ventral aspect of the right prepeduncle, where it bends to form the roof of the fourth ventricle, there was a very noticeable hemorrhagic extravasation, very dark, almost black in color, roughly quadrate in its outline, seven or eight millimetres (estimated) in its longest (caudo-cephalad) diameter, and about or nearly six millimetres transversely. Its mesal border closely approached the valvular margin of the peduncle but did not encroach upon it. Laterally the borders were well defined and straight, but caudad and cephalad it presented a ragged outline as if the blood had dissected its way irregularly among the fibres. There was no escape of blood into the ventricle and the lesion did not extend deeply into the tissues; the dorsal fibres of the peduncle were not involved. The right prepeduncle also showed one or two minute hemorrhagic spots on the ventral surface. There was no discoverable abnormality in either of the other cerebellar peduncles.

The brain, as a whole, without the dura, weighed forty-three ounces. The convolutions seemed to be of the usual type in arrangement. The brain drained very thoroughly in its removal from the skull, and no accurate measurement was made of the amount of sub-dural fluid; it was estimated however, to be rather in excess of the normal.²

In this case we have a capillary hemorrhage, involving only the ventral portion of the prepeduncle causing sudden disturbances of equilibrium and vomiting with, possibly, paresis on the opposite side of the body, if that was indicated by the statement of the attendants that the patient leaned heavily on the person supporting her on the left. The mental condition of the patient obscured many of the symptoms and nothing could be learned as to whether she

² I might state here, in addition that there was a very fluid condition of the blood generally; coagulation appeared to have been delayed or prevented.

suffered from headache, or other discomfort. It is probable indeed that her sensibility was generally blunted, and that she did not suffer pain as a normal individual would. There was no loss of consciousness for at least five hours after the first attack ; but, while there was evidence, or appearance rather, of some discomfort on the part of the patient there were no signs of any very severe pain or suffering.

The disturbance of equilibrium is altogether in accord with what is, I think, as much as any other, the accepted theory of the functions of the prepuncle, viz., that it contains the efferent fibres from the cerebellum, conveying to the hemispheres the impulses requisite for the finer coordinations. The statement of Stilling³ that it alone, of the three cerebellar peduncles, contains intra- and extra-ciliary and hemispherical fibres of the cerebellum, would suggest that possibly the functions of this part are more comprehensive than those of the others. However this may be, and assuming the possibility that it may have motor relations, indirect or otherwise, any contra-lateral paresis, occurring in the beginning of the attack, from a lesion limited to the prepuncle, is not easily explained on the supposition that the decussation of its fibres in the commissure in the pons is complete. The apparent paresis in this case would therefore seem to point to a partial decussation of the fibres, such as is stated to exist by Marchi,⁴ and which is admitted with a query by Spitzka.⁵

The lateral decubitus on the side of the lesion, and the drawing of the head to the right noticed in the beginning of the attack and lasting till the coma and relaxation had become general, are noteworthy since this has been described by Curschmann⁶ as a characteristic symptom of injuries of the prepuncle. It was not as lasting in this case as in the one described by him and no attempt was made to

³ Quoted by Wernicke, *Lehrb. d. Gehirnrk.*, I., 135.

⁴ *Gaz. degli Ospitali*, No. 67, 1886. *Jour. of Nerv. and Ment. Disease*, 1886, p. 719.

⁵ *Ref. Handb. of Med. Sciences*, viii., p. 186.

⁶ *Deutsch. Arch. f. Klin. Med.*, x'i., 3 and 4, 1873. *Schmidt Jhrb*, 162, p. 14.

alter the position to see if it would be reassumed, as was the case with his patient. The specially characteristic symptoms of lesion of the prepeduncle cannot be determined from clinical and pathological observations, as all of these in medical literature are complicated with involvement of other parts, but this deduction of Curschmann's from physiological experimentation, is not altogether unsupported by the present case. In this connection also, the rigidity of the arm observed later on recalls the experiments of Luciani on dogs, in which he observed after extirpation of the cerebellum a chronic contraction of the anterior limb.

The later appearing symptoms, the general relaxation and paralysis, and the coma, etc., preceding death were attributed during life to either an increasing clot causing pressure in the region of the fourth ventricle or to extensive ventricular effusion. The first of these did not exist, but I am inclined to think there was some abnormal intra-ventricular pressure. There was no obvious mechanical cause for this, and the effusion must have been due to a secondary irritative action induced in some way.

The cortical appearances of the hemispheres require mention here as they complicate the case to some extent. The conditions were not dissimilar to those met with in general paralysis, though there is generally a more recent and active meningitis in the latter, but there had never during life been observed anything pointing to such a diagnosis, either in the bodily or the mental symptoms. On the other hand, very similar appearances are occasionally met with in the autopsies of old terminal dements, or those who have been considered such for many years. It is not an impossible supposition that the brain was generally in such a condition that a congestive apoplectiform attack might have been excited by a sudden shock or injury of an important organ of the lower brain. I cannot think, however, if the cortical lesions played any part in the production of the symptoms observed in this case, that it was other than a secondary one.

ON THE GERM OF A COMMUNICABLE DISEASE
DERIVED FROM A DOG, ALLEGED TO
HAVE DIED OF RABIES, WHICH
RETAINS RABIES CHARACTERS.¹

BY RICHARD MOLLENHAUER, M. D.

SINCE an epizootic among dogs, claimed to have been rabies, was reported to exist in and around the County of Essex, in New Jersey, I have had several opportunities to witness or to verify experiments performed to test the legitimacy of that claim, and of certain statements which were made in connection with it.

I believe it is no longer necessary to criticize and expose the fallacy of the crude attempts to demonstrate rabies by introducing a fragment of brain tissue from a suspected animal under the dura mater in a trephined dog, the said fragment having been cadaveric from 24 to 48 hours. There seems to be little question that so much of Pasteur's statistics as was derived from a study of the so-called New-ark epidemic, is regarded by all, who are familiar with its history, as based on erroneous information.

In the course of my inquiries into the clinical and anatomical aspects of germ diseases, it occurred to me, that if the various forms of intense and attenuated virus employed by Pasteur were devoid of organisms demonstrable by our present methods of research, that we should be compelled to assume the existence of such organisms, or excluding them, to assume the infinite reproductive power of protozoans.

I had seen one human case, alleged to have been one of rabies, and as an enthusiastic and able bacteriological inves-

¹ Presented to the American Neurological Association in Philadelphia, June, 1890.

tigator found (?) and cultivated (?) the rabies microbe, notwithstanding his having had the specimen sent him in thin sections soaked in a solution of corrosive sublimate, whereas, neither microscopically nor by inoculation had I been able to obtain a convincing result from the *same material*, in a relatively *fresh* condition and untampered with, and as Fol is now generally regarded to have been misled in detecting similar features, I concluded to submit the whole question to as critical a test as circumstances permitted. As a result I may announce that the evidence I have of an existence in dogs of a disease resembling traditional rabies, is sufficient to justify my presenting it, —though there may be many gaps in the chain of evidence.

John C. Dancer, veterinary surgeon, at Orange, N. J., at considerable personal sacrifice, brought to the veterinary infirmary of Prof. James Hamill, D. V. S., a large spaniel.

The gentleman named, is one of the leading clinical experts in canine pathology ; he had seen one case many years before in the same neighborhood, presenting the same symptoms, and was in possession of good evidence of a fatal epizootic among dogs on an adjoining piece of ground. His opinion was thus expressed : " If this is not rabies, I know not how to class it." The dog was in a condition which may be described as intermediate to strychnine-tetanus and epileptic status. On his death, two dogs were inoculated—one by skull terebration, the other hypodermically—with the medulla emulsified with bouillon, under aseptic conditions.

The latter was alive and well up to May 21st, when he was utilized for another experiment. The former went raving mad—no other words can describe his condition—on the seventh day and died on the ninth day. The little animal dashed at everything and anything, bit and snapped at all objects within, and many out of his reach. On the seventh and eighth days he was absolutely fearless in his rage ; on the ninth day, he exhibited momentary obedience, or rather, was cowed by the lash, to break out again in fury after a few seconds. Like the first dog, he wished to drink,

and greedily lapped up fluids, but could not swallow them.

From this dog's parotid gland, from the œdematous tissue under the neck and jaw, from his blood, from his cortex cerebri and from his oblongata, cultures were made in bouillon-gelatine, and on potato, which yielded exactly the same results in both dogs, and from each of the organs named. After long and repeated efforts I succeeded in eliminating what I then thought were accidental contaminations, and retained two micro-organisms of which I have found *one* in every dog whom I succeeded in rendering rabid.

It would lead me too far, to detail the numerous failures which attended my experiments, until the last series, to be spoken of, had been reached. With the obtaining of a good series of oblique tubes filled with canine blood-serum, the bacillus of which I shall have to speak, first showed a healthy growth, and developed spores. It was the substitution of this material for that which had been used previously, which led me to anticipate that the disease here treated of, may yet be conveyed hypodermically.

Like most pathogenic germs, it possesses no great vitality, except in specially favorable soils. I am not committed to the view that we have to deal with a rabies as it is described by veterinary authorities. The character of the germ is such that it may turn out to be rather a facultative, than a pathogenic parasite. Be that as it may, the view of the freshly expressed parotid juice, swarming with these bacilli a few hours after death, in fact at the first moment of examination, gives rise to an anxious appreciation of the danger incurred from the bite of an animal, whose salivary glands contain such material. In connection with this reflection, I would refer to an older experience with a pathological saliva.

In the earliest series of experiments referred to, a spitz-dog having been trephined, and dying in convulsions, subsequently attributed to the meningitis which was found *post-mortem*—and to produce which had been the intention—some saliva was obtained and injected into a healthy dog, through the terebrated skull. He died in 36 hours ; and

from his case on, carried through seven generations, the parenchymatous fluids of successive dogs proved progressively more fatal, the last one dying in 8 hours after hypodermic inoculation.

I, unfortunately, at this time, was not prepared for extended bacteriological experiments, and owing to the absence of control inoculations, am unable to decide, even hypothetically, whether a ptomaine or a living germ had been a fatal agent of such unprecedented rapidity. The clinical symptoms and the gross anatomical appearances corresponded altogether to those found with death from rattle-snake bites.

When Spitzka demonstrated that the so-called characteristic symptoms of rabies could be produced by artificially provoked meningitis, he corrected an error so puerile that one almost hesitates to acknowledge that his laborious demonstrations were necessary to expose so glaring a fallacy.

My experiments, however, show that aseptic *cerebral* inoculations with a specific material, invariably kills in a period of from 6 1-2 to 9 1-2 days, and usually fails to kill when injected hypodermically (every part tried); yet no cerebral lesion competent to produce death, in fact no lesion, aside from the cerebral puncture can be found. One stubborn fact remains, and that is the great difference between cerebral and hypodermic inoculation with the same material.

How far cerebral inoculation may yet be regarded as a fallacious method, I am not now prepared to decide, nor even to surmise further than as regards the culture soil peculiarities of the brain.

The germ is a bacillus whose various growth-stages present a uniform type. In its adult period, it is usually found in chains typically made up of four, rarely of three, somewhat more frequently of two, and exceptionally of five links.

In artificial nutritive media the bacilli are much smaller than when found in the recently killed animal (examined in a hanging drop).

When first liberated from the spore, movements are seen sufficiently active, carry the bacillus after its "four link stage" over half the field given by a Leitz 1-12 immersion, in two hours, and in extra-organismal media, such movements continue feebly for twenty-four hours longer.

One end of the short chain, the one which is formed by the best developed link, is usually in advance and appears to be most active in motion. The other end of the link—particularly in specimens bred extra-organismal—is less bulky, more pointed, and bent slightly at its free end. The diagram will render clearer² than any description can, the relations and transitions of the phases found.

The various bacterial findings reported by European and American observers, have not stood the test of criticism; of one of them I have positive assurances convincing to my mind that not a mere error of observation but one of the crudest imaginable, can alone excuse the publications of a spurious discovery. Among the investigators who had devoted their labors to the discovery of the rabies germ, are some of the most competent bacteriologists. It is inconceivable that this germ could have been present in their specimens, undetected by them. From this and from other facts detailed in the full article, I deem it probable that rabic manifestations are symptoms of various diseases affecting the dog, some of which we know not the active agent of. And the surmise seems allowable that even a facultative parasite: the bacillus herein described if developed in stable soil, and accidentally taken up and propagated in the dog, may cause a disorder clinically undistinguishable from the rabies of veterinarians. The important practical outcome is that this large, easily identified and characteristic bacillus swarms in the parotid gland of the infected animal. There can be no doubt, if this chain of reasoning be sound, that under circumstances favoring its propagation, this germ may be introduced through the bite of the dog, and when successfully introduced, prove as invariably fatal as the traditional view regards true rabies.

² Owing to the great clearness of the microscopic picture, and the probability of obtaining good and clear micro-photographic pictures, their delineation is postponed.

ACUTE MYELITIS PRECEDED BY ACUTE OPTIC NEURITIS.¹

BY J. T. ESKRIDGE, M. D., DENVER, COL.

¹ Presented to the American Neurological Association at the Annual Meeting in Philadelphia, June, 1890.

THE subjoined case of myelitis is of interest chiefly on account of the rarity of the complication, or rather, the first observed neural disturbance, acute optic neuritis.

Charles B., aged forty-nine, engaged in railroad bridge building the last few years of his life, but formerly a stage driver in the mountain towns of Colorado, gave history of syphilitic infection about twenty years ago. He stated that there were two or three sores on the penis at the time, and that no constitutional symptoms followed. With the exception of the venereal trouble, he had always been in apparently perfect health until the beginning of his fatal illness. He had always indulged in alcohol freely, taking on an average, four to six drinks of whiskey daily, when he was where he could obtain them, but rarely drank to the point of intoxication.

On Jan. 1, 1889, he was feeling quite well, but noticed that after he had been looking at the sun's eclipse a short time, the vision of the left eye seemed defective. The next day there was a dull, heavy pain in this eye and it was increased by bright light. He did not seek medical advice at this time, but continued his work at bridge building. Two or three days later his vision became so poor in both eyes that he was compelled to discontinue his work. One week from the time that he had first noticed defective vision in the left eye he was totally blind in both eyes. At this time he consulted the railroad surgeon, Dr. Lay, of Denver, who has kindly given me many points in the history and to whom I am indebted for the privilege of studying and publishing the case.

Jan. 7th, when he first came under Dr. Lay's care, he he was seen by an oculist, Dr. E. C. Rivers, who found optic neuritis with white atrophy; vision in each eye, = 0.

There did not seem to be light perception. The next day (Jan. 8th), the right leg was observed to be weak and used awkwardly, but he walked from his boarding-house to the office of Dr. Lay and back, a distance of several blocks. On the following day (Jan. 9th), he was unable to leave his bed, as both legs were paretic. A few days later both legs were completely paralyzed, and about this time the sphincters of the bladder and anus began to be affected, and were soon completely paralyzed. Up to this time (about Jan. 15th), he had suffered no pain and had experienced no girdle sensation around the body. About Jan. 18th, he began to complain of a sensation as if there was a weight constantly lying on his chest, and found some difficulty in breathing. About Jan. 22d the arms became paretic, and he suffered pain, severe and shooting in character, in arms and in lower cervical and upper dorsal regions of the spine. The above history is as accurate as I was able to obtain it, as no records had been kept of the case prior to my seeing it.

On Jan. 26th, I was asked by Dr. Lay to see the patient in consultation with him. I learned from the patient that the night previous to my visit he had been able to see a bright light for the first time in more than two weeks. I inquired if he had ever been struck on the back, if his spine had been injured in any way; or if he had been subject to undue exposure, or lifting heavy weights previous to the onset of his trouble. His answers to my questions were all in the negative. No apparent cause, except possibly syphilis, was found for the myelitis.

Examination, Jan. 26, 1889:

The legs were swollen and incapable of the slightest voluntary motion, all their muscles thoroughly flaccid and the skin over them cool and death-like. Arms and hands retained but slight power of voluntary motion, the hands less than the arms, and the latter less than the shoulder muscles. The ulnar sides of the hands and fore-arms were weaker than the radials. Each hand registered, with the dynamometer, 3. All the abdominal muscles were paralyzed. The lower portion of the chest moved but little during respiration. No affection of the muscles of the face, tongue, pharynx or larynx. The sphincters of the bladder and anus completely paralyzed. All the reflexes, deep and superficial, in the trunk and extremities were absent. No response to the faradic current was obtained in the legs or arms. No galvanic battery was at hand, and consequently, no tests for reactions of degeneration were made.

In the legs, and in the trunk, as far up as the sixth intercostal space, all sensation was abolished.

He did not recognize the prick of a pin, the contact of hot and cold substances, the position of his legs, nor could he tell when the legs were moved. No sensation was felt when the catheter was passed into the bladder. On the chest, latterly and anteriorly, from the sixth to the third intercostal space he recognized the prick of a pin as painful, but could not feel the contact of smooth substances. Hot and cold substances were not distinguished as such, but were felt as painful, the cold causing the greater pain. From the third intercostal space upward, sensation was nearly normal, except in the second intercostal space, over which there was some hyperæsthesia. He was so weak that every movement of the chest and arms gave him such intense pain that I refrained from testing sensation over the back. The arms were (and have been for several days) the seat of a great deal of pain, sometimes dull, but usually sharp and lancinating in character. The pain prevented sleep unless it was relieved by morphia. The arms were not tender to the touch. There was considerable twitching of the muscles of the upper extremities. Movements of the arms, both voluntary and passive, increased the pain in the lower cervical and upper dorsal regions, and in the arms. The sense of touch was nearly or quite lost in the arms and hands, and with them he was unable to distinguish hot and cold substances as such. Ice or moderately cold water, he said, pained him, but did not feel cold. He felt the contact of hot cloths and warm metals, but said they did not feel warm, but gave a pleasant sensation. He felt the prick of a pin in the hands and arms, and could locate the position of the sharp points of the æsthesiometer, but he was unable to tell the position of either hand or arm. When I moved the hands or arms he could not tell in what direction I was carrying them, nor which arm I was moving. A large bed-sore had formed over the sacrum and was increasing in size, despite the utmost cleanliness of the parts. Wherever the skin over the legs was subjected to pressure, sores had begun to form. He had scratched himself over the breast opposite the fifth and sixth intercostal spaces, and the irritated parts had become considerably inflamed and showed no tendency to heal.

Special Senses.—Eyes : Light perception, with slight iritic reflex, was seen with each eye in the presence of a strong light. No ophthalmoscopic examination was made at the time on account of the extreme feebleness of the patient. Hearing, taste, and smell were preserved and about normal. The mind was perfectly clear and he had no head-

ache, and had complained of none since the beginning of the spinal trouble. Respirations were slowed at that time from the effects of morphia, under the influences of which it was necessary to keep him on account of pain. Pulse was 110; temperature, 99°. He continued to get weaker, and died about one week later (Feb. 3).

During the last week of his illness, the morning temperature varied from 98° to 99.5. Once it registered 103.2°. The evening temperature varied from 99.2° to 102°. The usual evening temperature was 100° or 101°. The day before, and the day of his death, it registered 103.2°, probably septic in character from the foul and sloughing bed-sores. The pulse varied from 100 to 140 per minute, and the respirations from 22 to 46.

Sectio-Cadavaris sixteen hours after death, by the writer, assisted by Drs. Fisk, Lyman and Weist.

The bed-sore over the sacrum was six inches in diameter, with ragged, sloughing edges, and at the centre extended down to bone, but no opening into the spinal canal was found. Small pressure sores existed over the buttocks, thighs, calves, and heels.

Cord.—The dura presented nearly a normal appearance, except opposite the sixth and seventh cervical and first dorsal vertebra, where it was thickened and inflamed. No pus was found except in two or three spots, each about one-quarter of an inch in diameter, on the outer surface of the membrane, opposite the sixth cervical vertebra. No bone disease was discovered. The pia was but slightly injected and did not seem abnormally adherent to the cord. The latter appeared slightly swollen, but to the touch was of nearly normal consistence. On section the cut surfaces were vascular and white, and gray matter were not clearly defined.

Brain.—The membranes and brain substance appeared normal throughout to careful macroscopic examination. The cord and its membranes, the optic nerves, chiasm and tracts, the great ganglia, the median portion of each occipital lobe, and the pons and medulla were placed in Muller's fluid.

Microscopical Examination.—The cord was fairly well

hardened, but the sections from the brain remained in the fluid too long and became granular. Stained sections from the lumbar, dorsal, and lower cervical regions of the cord showed marked inflammatory changes. The changes were diffused throughout nearly the entire cord substance, and not limited to certain portions of the cord, as we find in many cases of myelitis. In the upper cervical region and in the medulla the parts presented a normal appearance. Numerous micropic sections were made from portions of the brain, preserved in Müller's fluid, but the parts had become so granular that no satisfactory conclusion could be reached in regard to the existence of patches of intracerebral inflammation to account for the blindness.

Remarks.—From the paucity of literature on the subject, as well as from the writer's personal experience, the association of optic neuritis with myelitis is extremely infrequent.

Henry D. Noyes, M. D., in his "Treatise on Diseases of the Eye" (edition of 1884, p. 311), says: "Acute myelitis has, within a few years, been accompanied by affections of sight. The first case was published by Steffen and Erb, another by Dr. Seguin, of this city (New York), and I have joined Dr. Seguin in contributing another. The eye symptoms are those of acute, but moderate, neuritis optica, with remarkable impairment of the visual field and of central vision. There may be entire loss of direct sight, there may be any kind of irregularity in the fields, including total abolition on both sides or affection of one only; there may be repeated recoveries of sight and relapses. The peculiarity of the cases has been that vision, both direct and indirect, should undergo such great and unexpected variations. The lesion of the cord was in its lower and middle portions, as was fully manifested by symptoms of the bladder and the lower limbs. No explanation of the optic neuritis has been offered, although we may bear in mind that a root of the tractus has been traced by Stirling into the crus cerebri at the red nucleus of the 'hood' All the cases have gotten well, both in respect to sight and to the functions of the cord. In my own case, large doses of iodide of potassium

were employed, gradually reaching three hundred grains daily, and were well borne. The case occupied about four months in its evolution."

Seguin, in his *Opra Minora*, in an article entitled: "On the Coincidence of Optic Neuritis and Subacute Transverse Myelitis," discusses at some length, all the cases referred to by Noyes, and concludes as follows:

"In the spinal cord the inflammatory changes were in the dorsal region in all the cases, but in all other respects there were marked differences.

"In Case I., the right half of the spinal cord no doubt contained most of the lesions.

"In Case II., the æsthesodic region of the cord (posterior gray matter or peri-ependymal region?) was chiefly involved.

"In Case III., the entire structure of the cord must have been slightly affected, the motor region most. The comparative escape of the bladder in Case III. (no retention), is instructive anatomically, as the limitation of the numbness to the altitude of the region of the groin would indicate that the lesion was in the lowest dorsal or upper lumbar region of the cord, below the vesical centre. In Cases I. and III., where the limits of numbness and the constriction band indicated disease of the mid-dorsal portion of the cord, retention and cystitis occurred.

"The question naturally arises: Is there any causal or physiological relation between the two sets of phenomena observed in these three cases?

"Prof. Erb answers in the negative, and it seems to me that with our present knowledge of the relations between the optic apparatus and the spinal cord we must, in agreement with him, consider this association of optic neuritis and transverse myelitis as accidental."

Gowers, in his *Manual of Diseases of the Nervous System*, states: "In rare cases of myelitis, optic neuritis has been observed, without any intracranial complication to cause it." In a foot note he refers to a case, reported by Sharkey and Lawford (*Trans-Ophth. Soc.* 1883), and to one, observed by Dr. Dreschfield, of optic neuritis and dissem-

inated myelitis. The latter case terminated fatally. Whether or not the former did, is not so stated directly, but one naturally infers such a termination from the character of his reference to the case. Gowers remarks: "It is probably not the result of inflammation of the spinal cord, but is an associated and similar lesion, the result of the cause of the myelitis. It is noteworthy that most of the cases thus accompanied have been instances of disseminated myelitis, a form that suggests a cause acting widely on the nervous system. In the case of Sharkey and Lawford the optic neuritis reached its height some weeks before the occurrence of the first spinal symptoms, and in the cord were two separate and distinct foci of inflammation."

The case, a detailed report of which is found in this paper, differs from the one reported by Erb, and from the two described by Seguin in his *Opra Minora*, in that it was a severe case of acute myelitis terminating fatally in five weeks from the first appearance of eye-symptoms, and that the optic neuritis was decided and well-marked when first seen, no vision, except slight light perception, returning subsequently. It differs from those cases referred to by Gowers, as observed by Sharkey and Lawford, and by Dreschfield, in that there was diffuse ascending myelitis and not disseminated as those cases seem to have been.

There is one curious noteworthy fact observed in several of the reported cases, as well as in the one which forms the subject of this communication, viz: the myelitis began in the lower portion of the cord. In the writer's case the inflammation began in the extreme lower portion and gradually travelled upward. In Dreschfield's case, and in the one under discussion, the optic neuritis reached its height before any cord symptoms were manifest, in the former the interval being two weeks, in the latter case, a few days.

It is useless to speculate, in the absence of further observations, as to the cause of optic neuritis in association with acute myelitis. The writer can only regret that through his negligence he allowed the specimens taken from the brain to remain in Müller's fluid so long that they were unfit for reliable microscopical examination.

Periscope.

THE STUDY OF CEREBRAL HEMIPLEGIA.

. Wernicke ("Berlin. klin. Wochenschrift," 1889, No. 45) calls attention to the clinical signs of hemiplegia, which heretofore have not been observed nor their value recognized. Corresponding to the well-known fact that, in the facial paralysis accompanying hemiplegia, the orbital portion of the nerve is usually unaffected, a similar phenomenon may be observed in the upper extremity in the fibres of the spinal accessory nerve. This nerve divides into two branches—one supplying the sterno-cleido-mastoid muscle, and the other the trapezius.

As a rule, the branch to the sterno-cleido-mastoid escapes, while that supplying the trapezius is paralyzed. The latter paralysis manifests itself in the drooping of the shoulder while at rest.

The paralysis in the lower extremity, which is usually neither complete nor permanent, also shows several characteristic peculiarities, which explains the fact that walking is still possible, even in severe cases.

In such patients, while in the supine position, one can demonstrate that active elevation, to a certain height, of the extended leg can yet be accomplished, although feebly.

The dorsal flexion of the ankle-joint is nearly or completely abolished, but plantar flexion can be performed with considerable force.

In the prone position the flexors of the knee-joint are nearly or completely paralyzed, while the extensors show a well-marked or almost normal strength.

Therefore, in *hemiplegia*, the muscles which are especially important in locomotion are the ones that are the least affected.

W. M. L.

A CONTRIBUTION TO THE PATHOLOGY OF TABES DORSALIS.

E. Adamük ("Archiv f. Augenheilkunde," Bd. 20, p. 307) reports a case of tabes occurring in a girl, eighteen years of

age, in which the disease terminated fatally at the end of nine years.

Onanism was the only etiological factor that could be accepted, syphilis and exposure to cold being excluded. The disease began with ataxia, lancinating pains, and paræsthesiæ.

In the third year the sight became affected, which progressed until typical optic nerve-atrophy was developed with almost complete blindness.

The autopsy revealed the picture of *sclérose en plaque*. Round sclerotic patches were found distributed over the entire cortex of the cerebrum and cerebellum and in the medulla oblongata and spinal cord. The cranial nerves were involved in a similar manner, especially the optic and the third nerves. The view is expressed that this case favors Förster's theory of the origin of tabes; that in this affection the same histological changes may take place simultaneously or successively at different points in the central nervous system, which seems especially predisposed thereto.

W. M. L.

THE EXCESSIVE USE OF COFFEE, AND ITS DELETERIOUS RESULTS.

F. Mendel ("Berlin. klin. Wochenschrift," 1889, No. 40) maintains that caffein, even in the form of the customary infusion, when it has been used in excess for some time, is capable of producing a typical clinical picture.

The symptom group which is thus produced is characterized by disturbances of the central nervous system, and by its deleterious effects upon the muscular and circulatory apparatus. In support of these views, the author has noted a feeling of general malaise, indisposition for work, despondency, nervous exhaustion, and cerebral neurasthenia.

The action on the muscles manifested itself in a considerable diminution of motor power, which was especially noticeable in movements attended with exertion. There was also tremor affecting the hands. Moreover, the pulse was weak, accelerated, and irregular, and the heart-beat feeble. Sensations of anxiety and cardiac palpitation also occurred.

Disturbances in the alimentary tract were also observed, such as hæmorrhoids, obstinate constipation, and nervous dyspepsia.

As the proper plan of treatment, he recommends the

complete abstinence from the use of coffee; the avoidance of all excessive exertion; rest and nutritious food.

As a prophylactic measure, excessive indulgence in coffee should be discouraged and its use interdicted in all long-continued diseases accompanied by fever, such as typhoid fever, tuberculosis, chronic pyæmia, in chronic diseases of the brain and spinal cord, in disturbances of the circulation, and in all diseases caused by defective hæmatosis.

W. M. L.

THE PATHOGENY OF VICE.

A series of interesting papers on this subject has appeared in recent issues of the "Western Medical Reporter." One influence that promotes crime is the system of herding together of all sorts and conditions of criminals in reformatory (?) institutions. Among such evil associates the lad committed for petty larceny easily acquires the art of burglary while in prison. One youth stated he would never have been in Sing Sing except for earlier incarceration in a House of Refuge, which he described as a perfect college of crime.

Homicidal impulses result from disease. The epileptic is often a murderer simply because he has epilepsy. A lack of will-power is often due to the action of narcotics upon the brain—alcohol, absinthe, opium, chloral, hasheesh, and cocaine; and "what's in a man when he's sober comes out when he is drunk."

The more lowly organized the brain, the more nearly then its possessor approximates the brute creation. After sunstroke, brain-cells have been left so impaired that alcohol sometimes turns the sufferer into a raving maniac who is impelled to violence. Such a man needs seclusion and treatment, not punishment.

The repression of prostitution the author considers too comprehensive a subject for present discussion, its moral, sociological, and physical aspects requiring the most careful attention. At the same time, he gives certain headings that afford food for thought:

I. The duty of parents in regard to instructing children in matters of sex and reproduction. [This can be done easily by means of plants first, then the lower animals, then the higher, and, lastly, humans. Remember always that Dumas is right when he says children are as old as their questions—that is, their persistent questions.—L. F. B.]

II. The butterfly element in society, which demands that young men and young women shall live and dress more luxuriously than their means will permit, and hence the firm belief that matrimony is the prerogative of the wealthy few.

III. The partiality of the world for men, and consequent double standard of morality that presents striking unfairness in regard to the misguided or vicious conduct of women.

IV. The peculiar commercial cupidity which demands that dependent women shall work for almost nothing and support themselves on such salaries. The responsibility which is daily assumed by the proprietors of wealthy commercial enterprises is something to make one reflect upon man's inhumanity to woman.

V. Diseased conditions and hereditary traits in women.

It will be perceived that vice needs a new classification. And when all kinds of viciousness has assumed in the public mind its own local habitation and name, the word *blame* will gradually fall into disuse, and the criminal will become the joint ward of the doctor, the philanthropist, and the financier.

L. F. B.

NUTRITION IN HYSTERIA.

Two physician's in Charcot's clinic, Gilles De La Tourrette and Cathelineau, have undertaken the study of this subject. Their observations appear in the "Progrès Médical." In spite of Empereur's dictum that "mal-assimilation does not occur in hysteria," the authors undertake to show, by varied and careful research, how constant is impaired nutrition and how rapid the change for the better as the patient recovers from the attack. Weight increases in the same ratio that it was lost. Excretions are greatly diminished. Were it not for this fact the loss in weight would probably be even greater than it is.

L. F. B.

URIC ACID AND PERSONAL PECULIARITY.

Dr. George Harley, in "Lancet," April 5, 1890, states—while correcting a typographical error, in the discussion following Sir Wm. Roberts' paper, which transforms "birds" into "bears"—that he has endeavored to show the fallacy of supposing that diet exercises a greater influence on the

production and excretion of uric acid by the human body than constitutional peculiarity. Within the first month after its birth, the human infant excretes a proportionally larger amount of hippuric than of uric acid. The sucking calf's urine contains no hippuric acid at all. Both are milk-fed, and the slight difference in their mother's milk cannot be said to be sufficient to account for this difference in the nature of their urinary crystallizable excrementitious acid ducts. Sugar is supposed to augment the formation of uric acid in the human body. Yet experiment proves that until the digestive functions become seriously deranged there is no evidence of increase of uric acid in the urine. Under certain pathological conditions men have been known, even when many days on a spare diet, to eliminate enormous quantities of uric acid. Muscular exercise increases and hot weather diminishes the excretion of uric acid by the urine. Nevertheless, the following biological facts totally upset the validity of the theory based upon these data. The slow-breathing, sluggish-moving, carnivorous serpent, and the quick-breathing, muscularly active grain-eating bird, and the more strictly vegetarian insect—as the bee and the butterfly—all pass uric acid in their solid urines. And that, too, as far as has yet been ascertained, in much about the same relative proportions to their bodily weight. Hence, the argument that constitutional peculiarity, both as regards species and individuals, is a much more important factor in the production of uric acid in the animal economy than mere diet. L. F. B.

FORCIBLE FLEXION OF THE BODY IN LOCOMOTOR ATAXIA.

The "Canadian Practitioner," January 1, 1890, quoting from another journal, states that at the thirteenth Congress of the Italian Medical Association held at Padua, Dr. P. Bonuzzi communicated the results of a number of experiments made on the cadaver, with the view of ascertaining the physical effects produced on the spinal cord by suspension. During suspension the spinal cord is displaced upward from three to four millimetres, this resulting from slight increase in the distance between the vertebræ owing to relaxation of the muscles and stretching of the vertebral ligaments. The roots of the spinal nerves, with the exception of the cauda equina, do not seem to be appreciably stretched, although they are slightly altered in position. The tension of the cerebro-spinal fluid is increased. During suspension the vertebral column is apparently lengthened

to the extent of from one and one-half to three centimetres. This elongation is due more to separation of the spinous processes than to separation of the bodies of the vertebræ. The body as a whole is lengthened during the process of suspension from two to three centimetres. In a second series of experiments Dr. Bonuzzi found that by bending the body forcibly forward so as to bring the knees in contact with the abdomen, the spinal cord and cauda equina are subjected to considerable stretching. Having made an opening into the vertebral column and inserted a needle perpendicularly to the long axis of the cord, he noted that on bending the body forcibly forward, the needle was carried downwards for a distance from eight to twelve millimetres, the spinal cord becoming thinner and more resistant, the cauda equina being extremely tense. Traction on the sciatic nerve stretched the cauda equina, but did not draw down the cord more than two millimetres. Progressive and lasting improvement resulted from forced flexions in the case of a woman suffering from locomotor ataxia, thus securing the advantages of suspension without its drawbacks.

L. F. B.

THE WARNINGS OF GENERAL PARESIS OF THE INSANE.

In the "British Medical Journal," April 5, 1890, is Dr. George H. Savage's paper with the above title. Extreme difficulty is often found in distinguishing between causes and early symptoms; for in many cases what may be causes of general paresis, in others may be signs of the disease. Possibly drink, extravagance, and sexual excess alone or combined may start the degenerative process; but it is also certain that each or every one of these may be early signs of loss of the highest self-control.

General paralysis is a degeneration rather than a specific disease, which is most commonly met with in middle-aged married men, inhabitants of cities, flesh-eaters, and drinkers of alcohol. It is not common among the congenitally deficient or among epileptics. It is a frequent follower of constitutional syphilis, especially if this disease has affected the higher nervous organs or their envelopes, and is not uncommonly related to head injury or to causes of nerve-tissue disease, such as those produced by lead. A feeling of fatigue is an important early symptom, associated with indecision, doubt, and a tendency to look on the dark side of things, or even hypochondriacal weakness. It may precede other symptoms by a year or so, and be replaced for a

time by a morbid bouyancy. This condition is only valuable as a symptom in connection with others, inequality of the pupils, loss of power of expression by speech or writing, etc. Ataxic symptoms may precede general paresis by an almost indefinite period, and may be its first manifestation, in which case syphilis is included in the history. Temporary aphasia is another important warning symptom. Alteration in handwriting is another sign of moment. Some patients give up writing, or alter their mode of holding the pen, for a year or more before showing other symptoms of general paresis. Facial expresion is early affected. Friends say the patient has developed a "fat face," for obliteration of lines give an aspect of fatness. At the same time the skin may become greasy, or there may be unilateral sweating. Ptosis and external strabismus are rare as symptoms of general paresis; but they are common as expressions of a morbid process that ends in general paresis. Allied to aphasia are slight and partial losses of power or sensation. Such attacks are usually related to slight fainting fits. Headache and facial neuralgia are the most common forms of pain preceding general paresis. Generally headache is rare among the insane. Double sciatica is a symptom not to be overlooked. The optic discs may give much information. Impairment in the sense of hearing, taste, and smell are not uncommon.

The gradual loss of power of social accommodation is one of the earliest and most marked of warnings. Stupid stealing and thoughtless indecency are noticeable defects. Changes of temper and character precede general paresis. The sudden outbreak of mania is a precursor of this disease, as may be also epileptoid seizures at irregular intervals, and hysterical or hysterio-epileptic fits. General paresis has a local origin in many cases, and the surgeon may yet scrape out necrosed brain as necrosed bone is now treated. But there is small measure of hopefulness possible, for degeneration follows on the predisposing condition. The fungi grow on the dung-heap, but they do not form it. L F.B.

DOUBLE ATHETOSIS.

The "*Progrès Médical*," January 18, 1890, contains a notice of an Italian work on this subject by Roberto Mas-salongo. The author's researches have convinced him that only a limited number of cases have been reported: ten in France, four in England, eight in Germany, and nine in America. Four have come under his own personal obser-

vation, and the records of these are the first published in Italy. A *résumé* of other physicians' study of this condition is given, and his own cases given in detail, together with the differential diagnosis of disseminated sclerosis, locomotor ataxia, Friedreich's disease, and ordinary chorea. Three of his cases were children of the same parents. A view of the facts collected in regard to the distinctive characteristics of unilateral athetosis or hemiathetosis, and double athetosis:

Hemiathetosis.

1. Only one side of the body is affected.
2. In the majority of cases the affected side is more or less hemiplegic.
3. Hemiplegia precedes the athetosis.
4. Usually there is more or less anesthesia on the affected side.
5. In repose, the movements are more violent and persistent. The facial muscles are but rarely implicated.
6. The autopsy has always revealed profound intracranial lesions.
7. Hemiathetosis is not an independent affection, but secondary to well-known cerebral lesions (the posterior half of the internal capsule, etc.), as is hemichorea.
8. Hemiathetosis finds its analogue in symptomatic hemichorea.

Double Athetosis.

1. Both sides affected.
2. Double athetosis is most frequently found among idiots and imbeciles.
3. Is primary, congenital, or appears in early childhood.
4. Is not preceded by motor paralysis, and sensibility is ordinarily preserved.
5. The movements are less violent and persistent in repose. Facial muscles and even muscles of the tongue are frequently implicated.
6. In the only two cases where an autopsy has been performed, lesions of the dura mater and of the convolutions existed.
7. Double athetosis is a special independent, and primary affection.
8. Double athetosis finds its analogue in common chorea.

L.F.B.

ATAXIC PARA-PARESIS.

Dr. Henry Lyman in ("The Medical and Surgical Reporter," March 29, 1890, consider this subject). The patient, sixty years old, began, two years before, to have pains in different parts of the body, which have continued to the present time. Walking in the dark is difficult; the Romberg-Brauch symptom present, together with exaggerated patellar tendon-reflex, paresis of the ocular muscles, some disturbances of sensation, and a slight ankle clonus on the right side.

Why is this not locomotor ataxia? Because there is no loss of patellar tendon-reflex; neither is there lancinating pain. The disease under consideration is one of degeneration, not of inflammation. Gowers considers it a disorder

by itself, and calls it ataxic paraplegia. It is ataxic, but not exactly a paraplegia. This term should be reserved for those cases in which there is complete loss of power. Ataxic para-paresis answers well when there is apparently a chronic weakness in the lower extremities, increased knee-jerk, etc.

There is no syphilitic history. This is true, probably, of all instances of ataxic para-paresis. Locomotor ataxia has its seat in the posterior columns of the cord. In this case to some extent this is also true; but principally the lateral columns are affected. It is a disorder, apparently, in which there is a tendency to generalization of the degeneration through the pyramidal tracts of the cord and to invasion of the posterior columns.

If the disease happens to strike the central portions, near the median fissure, remote from the posterior nerve-roots, then the patient does not complain of much pain. If the nerve-roots are encroached upon, there is pain, as in locomotor ataxia. The course and treatment of ataxic para-paresis are identical with that of tabes dorsalis. L. F. B.

SECONDARY IDIOCY.

(*Ibid.*) After any infectious disease there may be secondary lesions that impair the mind. Especially is this true of syphilis; also of typhoid fever and diphtheria. This child, eight years old, is a case of mental destruction following diphtheria. She is strong, eats well, and is perfectly quiet at night. The mental disturbance manifests itself by irritability, running from one end of the room to the other, throwing the head from side to side, and pounding the top of the head with the hand. When hungry she becomes angry, cries, but is quiet when something to eat is secured. Were the child's condition congenerital, the outlook would be more hopeful. There has been a destruction of what existed, and there are no means at medical command to replace it.

L. F. B.

Society Reports.

AMERICAN NEUROLOGICAL ASSOCIATION.

*Sixteenth Annual Meeting, held at Philadelphia, June 4th,
5th, and 6th, 1890.*

(CONTINUED.)

Dr. W. R. BIRDSALL, of New York, read a paper, entitled "A Comparison of the Value of Different Therapeutic Methods Employed in the Treatment of Nervous Diseases." He first called attention to the small number of papers on therapeutic subjects which had been read before the Association since its commencement, and to the relative barrenness of neurological literature, in all languages, respecting therapeutic contributions. He referred to a prevailing impression among many of the profession that notwithstanding the vast amount of knowledge possessed by the neurologist concerning the anatomy, physiology and pathology of the nervous system, and his skill in the diagnosis and localization of disease, his ability to cure disease was extremely limited. Dr. Birdsall, while admitting that many of the diseases which the neurologist is called upon to investigate are practically incurable, maintained that those who saw no advance in the therapeutics of nervous diseases were probably looking in the wrong direction for progress, the advance being in great part the outcome of those very investigations considered by many as impractical scientific refinements. The early diagnosis of disease he regarded as the most important factor for therapeutic success in diseases of the nervous system, as it frequently enabled the physician to check the course of a disease at a stage when marked disability had not yet resulted. The measures to be chiefly relied upon for such an arrest of disease were hygienic, rather than pharmaceutical. The key to the progress we are making being early recognition of disease and adaptation of the patient's ways of living to his changed conditions. He did

not wish to be considered as one who failed to appreciate the value of drugs, for he believed they were indispensable accessories in the treatment of most diseases. His object was, rather, to lay stress on the general lack of appreciation of hygienic therapeutics, and particularly on the frequent neglect to enforce hygienic measures in detail. He called attention to some of the difficulties of the hygienic plan of treatment, extra time and care being required to study patients so as to properly direct their habits. General rules not being sufficient, specific details were necessary for each individual; failure in this regard accounting for much of the unsuccessful work in our hygienic recommendations; still another obstacle being the inability to enforce obedience on the part of the patient. Faith in some drug whose properties were unknown, confidence in some measure not understood commanding obedience in execution not easily secured when the details of treatment consisted of well-known acts modified to adapt the individual to his disturbed condition. He laid great stress on the personal power of the physician to enforce his rules when he has confidence in them, but thought that too often the physician erred from over-confidence in his drugs, the neglect of hygienic measures rendering his medicines inoperative, while they might have served as useful accessories had he not relied upon them to do all. He believed that knowledge on the part of the patient of what is best or right was a weak power compared with the personal force of command which a physician can usually exert over his patients; and that physical morality would be more permanently organized in this manner than by any amount of ideal moralizing. He then considered some of the essentials of hygienic therapeutics. In diseased states the work put upon the different organs should be adapted to their enfeebled powers. All relations between storage and expenditure of energy must be readjusted to the disturbance in equilibrium, and the art of therapeutics consisted in bringing about such a readjustment by any means in our power. Careful regulation of the expenditure of energy was the most important factor in the problem. He called attention to the importance of rest in certain diseases, of the great care required in regulating the amount and kind of mental and physical work done in degenerative affections of the nervous system and also in functional nervous disorders. While rest from overwork was essential in some diseases, in another class the lack of balance between physical and mental work was the faulty condition. The activities of the muscles, the skin

and the lungs being allowed to wain, the overworked nervous system becomes impoverished through over-expenditure, a deficient nutritive supply and defective excretion. The regulation of gymnastic work he regarded as an exceedingly important, though a most difficult task. The modern craze for so-called physical culture he believed was bringing forth dangers as great as those it is sought to remedy through over-training, improper training, training for brain workers which fatigue rather than rest the brain, together with other faulty methods. Questions of diet were briefly considered, and then followed a review of other therapeutic methods than the hygienic. The agents heat, cold and electricity were considered. Hydrotherapy he believed to be as much neglected in this country as it was overdone in Germany. Electrotherapy he thought was overestimated by many enthusiasts, but undervalued by its opponents. Electricity served special purposes for which it was superior to any other therapeutic agent, but it should not be considered a cure-all. Cutaneous irritation he regarded as the most important therapeutic measure which we possess, next to hygienic therapeutics; constituting the chief therapeutic factor in the use of the agents previously mentioned. The value of sinapisms, blisters, and the cautery, of cupping, massage, and vibratory therapeutics, were considered in this connection. Surgical therapeutics to nervous diseases were referred to, including the modern surgery of the brain and spinal cord. Finally, the drugs usually employed by neurologists were briefly reviewed, and the conservative position of most writers favorably commented upon in that they recommend them for symptomatic indications rather than with the expectation of curing the morbid process in most conditions. Dr. Birdsall thought that the recent accessions to our list of new remedies should make us hopeful for the future. Though the great field of hygienic therapeutics must be our chief reliance, yet we must palliate, and temporize, and we should experiment; but we should treat other therapeutic agents as accessories, seek to value them for what they are worth, and recognize their limitations. The treatment of the principal diseases of the nervous system was then reviewed and a comparison made of the different therapeutic methods usually employed in each.

DISCUSSION.

Dr. SINKLER judged that the author of the paper was pessimistic and nihilistic in the matter of drugs. For him-

self he admitted having a belief in their utility in nervous diseases, though he feared it might be an evidence of low mental calibre. Dr. Birdsall had referred to arsenic in chorea. There was no doubt that hyoscyamus, and its alkaloids, in tremors of various kinds, and the iodides and antisyphilitic remedies in specific diseases of the nervous system, were each of value. He simply wished to place himself on record as being neither a pessimist or nihilist.

Dr. BUSH said he should like to accentuate what Dr. Birdsall had said as to the general management of cases. A great many cases of nervous disease could be better handled other than by drugs. He should at some future time present some cases of insanity from drug poisoning. The question usually addressed to him by visitors was: "How do you put patients to sleep?" People seemed to expect the existence of some unusual hypnotic. Time was when he used to try everything that came along. Now he used drugs less and less. The majority of patients could be better managed by baths, moderate exercise, quiet, diet, and attention to the alimentary tract generally than by drugs.

Dr. GRAY thought that Dr. Birdsall's paper was very opportune, because there was no question but the tendency existed among neurologists to spread the idea that there was no therapy in neurological science. Nervous diseases were supposed to be self-limited or incurable. It was his custom to make liberal use of drugs, in combination with other means, in treatment. Chorea, in most of its minor forms, neuralgia, functional nervous diseases, not acute or acute mental disorders, except acute mania, were all amenable for amelioration or cure by the judicious employment, with other means, of properly directed therapeutic remedies. He would like to protest against the stand that had been taken in opposition to electricity.

PONS LESIONS IN THEIR RELATION TO ASSOCIATED EYE MOVEMENT PARALYSIS.

Dr. SPITZKA presented this paper by title, accompanying the same by demonstration of a specimen of a minute focal lesion of the dorso-caudal part of the pons, unilaterally situated in and near the abducens. In connection therewith Dr. Putnam had mentioned one of similar location to Dr. Spitzka's, in that the presumable lesion, for the patient had recovered, must have had a location similar to the one in the former case.

Asylum Notes.

THE NEED OF ASYLUMS FOR INEBRIATES.

BY T. D. GROTHERS, M.D., HARTFORD, CONN.

While theories of the nature of inebriety may differ, the actual necessity and practical character of some special form of restraint for the inebriate is conceded beyond all question. The restraint in the station-house and jail fails to do more than bring temporary relief at the expense of making the case worse. The insane asylum fails, and its work for the insane is impaired by the efforts to help the inebriate. While they are of the same family types of disease, they require very different conditions of care and restraint. The inebriate who persists in poisoning himself with alcohol or other narcotics drugs is certainly a madman, and should be restrained and prevented from suiciding himself and family. He is a source of danger which should be recognized and prevented as much so as poisoned water supplies, or infectious germs. In almost every community cases can be found of inebriates who are destroying themselves, their families, and fortunes, and no effort is made to stop them ; unless they commit some crime or violate some statute law. An asylum for this class and a law giving power to restrain them for a sufficient length of time to give reasonable hope of recovery would be felt in every neighborhood. Several private asylums for these cases furnish data, showing the possibility of cure in a certain proportion of these cases. An institution that would combine separate and distinct features in the surroundings and means to both restrain the inebriate and treat his diseased impulses would be no experiment or doubtful work. The license fund from the sale of spirits should be appropriated for the building of asylums or workhouse hospitals in the country for this class. Some kind of manual labor should be a means of treatment where it could be used to the advantage of the case. The inebriate should be isolated and housed in such places, not as a punishment for his trouble, but as a sanitary measure by the community to protect the person from himself, and his family from destitution, and the society from the damage which follows a reckless course of drinking. In this way many sources of insanity, criminality, pauperism, and disease could be greatly lessened.

The increasing number of inebriates and the direct and indirect injuries which follow from them demand a medical recognition and study of these cases. Special hospitals are called for to not only to quarantine them, but to place them in the best possible conditions for temperate living and health. The two classes should come under medical care. The recent and curable cases and the chronic and incurables, both should be separated in asylums and hospitals,

and come under absolute restraint, rest, building up and training for a future of temperate living. In the recent cases, after the rest and restraint of hospital care, many of them would be able to go back to active life again. In the incurable cases they could be protected housed, and in some degree made self-supporting by the industrial hospital work. These hospitals are already demonstrated successes in many ways. All that is needed at present is a united medical interest to pass laws giving full power of control over these cases, and a full appreciation that this great question is a medical and sanitary one, which cannot be understood or treated from any other than a scientific point of view. Inebriate asylums to set apart and control these suicidal drug maniacs is the first most practical measure. This is the real temperance work which physicians should urge. We must recognize that these cases are dangerous, not only to themselves and families, but to the community in which they live. All such cases forfeit their right to freedom and liberty, and all rules of common sense and self-protection, demand that they should be put under the care and control of others in special hospitals. Each town and city could have these special hospitals without additional cost to the taxpayers by making the taxes on the spirit dealers support them. This could be justly done on the principle, that all special trades should protect and take care of the accidents which follow from them. Thus railroad accidents entail responsibility on the railroad companies, and the dealer and manufacturer of spirits should be held responsible for the victims of drink.

In 1864 the first asylum for inebriates in the world was opened at Binghamton, N. Y. This was closed fourteen years later, but not until it had demonstrated to the world that asylums of this class were eminently practical. To-day there are one hundred asylums in existence in the world, and it would seem as if their need and value was firmly fixed. Yet the medical profession has not taken any general step in this direction. The need of asylums for the care of inebriates is the most urgent of any medical work of to-day, and one that gives the largest promise of success.

CORRECTION.

In the August issue of this JOURNAL an omission occurred in the description of Fig. 1, in the article by Dr. McBride. As corrected, the description should read as follows, the words in italics being omitted in the original description :

Fig. 1. Line shows approximate depth of tumor. Shaded portion shows superficial destruction of brain-substance. Arrow shows starting-point of *perpendicular section*, as shown in Fig. 2.

THE
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Nervous and Mental Disease.

Original Articles.

A CONTRIBUTION TO THE PATHOLOGY OF
SOLITARY TUBERCLE OF THE
SPINAL CORD.*

BY CHRISTIAN A. HERTER, M.D., NEW YORK.

THE exceedingly infrequent occurrence of massive or solitary tubercle within the substance of the spinal cord, and the difficulties which at present attend the recognition of the condition during life have rendered its study of comparatively little practical importance. Nevertheless there is reason to hope that some of the difficulties in diagnosis which now exist may be gradually removed by a careful study of the cases that from time to time occur and of those that have been reported, and it is with such a possibility in mind that this paper has been prepared.

The paper consists of a report on the clinical history and pathological anatomy of three hitherto unpublished cases of solitary tubercle of the cord, and of a brief analysis of the clinical and pathological features of the condition based on these cases and on those which have been collected from the literature.

The following is a report of the new cases. The clinical histories of these cases are in some respects incomplete, but

* Read at the annual meeting of the American Neurological Association held in Philadelphia, June 4, 1890.

not sufficiently so to destroy their value for the present purpose.

CASE I.—W. S., aged twenty-eight. Waiter. Admitted to Dr. Ball's service at St. Luke's Hospital January 3d, 1890. Gives family history of phthisis, mother and two maternal aunts having died of this disease. Also gives usual personal history of pulmonary phthisis, dating back about one year. About three months ago the patient developed tubercular epididymitis of right testicle.

At time of admission he was anæmic and emaciated and a physical examination revealed the ordinary signs of tubercular disease at the apex of either lung.

Two weeks before admission the patient was able to walk without difficulty and had no symptoms suggestive of organic disease of the nervous system. Headache, it is true, was present much of the time, but was not of the character to lead to a suspicion of intra-cranial disease. Apparently the first symptoms of spinal cord disease were spasmodic pain and occasional rigidity in the lower extremities. Then there developed rather rapidly weakness of the left leg and hyperalgesia of both legs. Very soon the weakness extended to the right leg and the hyperalgesia became decided and constant.

Occasional attacks of severe pain occurred as time advanced. The motor paralysis grew rather rapidly worse, but the loss of power continued more marked in the left leg. Anæsthesia of the lower extremities was not noted, but may have been present.

An examination a few weeks before death showed that the knee-jerks were very active and that ankle-clonus could be obtained on either side.

A few days before death the patient complained of a girdle-pain about the waist.

On Feb. 15th, not quite six weeks after admission to the medical wards of the hospital, the patient died in stupor. For several days before his death he was semi-comatose or delirious, and had a high temperature. Toward the end the paralysis of the left leg was all but absolute.

The autopsy, by Dr. Thatcher², showed the existence of wide-spread tubercular disease. The lungs were the seat of numerous clusters of miliary tubercles, which were located

² I am indebted to Dr. Thatcher both for the notes of the autopsy and the specimen.

chiefly in the upper lobes, and contained a few small cheesy nodules.

The bronchial glands were enlarged and cheesy. The omentum showed numerous adhesions. The mesenteric glands were prominent and a little cheesy. The intestines were the seat of numerous ulcers.

The kidneys and ureters were free from tubercular disease, but the left supra-renal capsule was almost entirely replaced by a cheesy mass. The tunica vaginalis of the right testicle was adherent, and both epididymis and testicle were enlarged and cheesy.

The prostate was enlarged and contained a cheesy mass in its left lobe. The seminal vesicle and commencement of the vas deferens on the left side were cheesy. The brain was the seat of several massive tubercles. Of these, three were in the cerebellum. One of them, about one-half inch in diameter, was in the posterior edge of the left cerebellar hemisphere. This nodule extended to within an inch of the posterior median fissure and to within a quarter-inch of the posterior surface of the cerebellum.

Another, somewhat larger than the first, was situated almost symmetrically with it in the right cerebellar hemisphere. It reached into the cortex of the cerebellum. Just below and external to this nodule was another of smaller size. Another cheesy mass of the same character occupied the anterior extremity of the right optic thalamus. Along the fissure of Sylvius was a yellowish exudation into the pia.

In the 7th and 8th dorsal segments of the spinal cord was a rounded cheesy nodule with a grayish border. This nodule was about three-quarters of an inch in vertical extent. At the level of its greatest diameter the mass occupied almost the entire area of the left half of the cord, extending to within one-sixteenth of an inch of its periphery in front and behind. The right side of the cord was also encroached upon to a considerable extent, but a large part of its white substance, especially that of the lateral columns, was intact. Just above and below the tumor the cord was slightly softer than normal. When the cord was har-

dened in Muller's fluid, secondary degenerative changes were observed in certain white tracts above and below the mass. Below the tubercle the crossed pyramidal tracts of either side were degenerated, the appearance indicating that the process was a little more advanced on the side of the tumor than on the opposite side. Above the tubercle the changes were limited throughout their extent to the posterior median columns, neither the direct cerebellar tract nor the column of Gowers appearing to be implicated.

The position of the degenerated areas in the posterior columns was unusual and deserves brief mention. In the dorsal region, just above the tumor, the degeneration was confined to a small strip on either side of the posterior median fissure at its anterior extremity. These strips were oblong in contour, lay close to the fissure, and extended but a short distance back from the posterior commissure. The degenerated area was larger and more distinct at this level on the left side, *i.e.*, on the side most invaded by the tumor. In the upper dorsal region the degenerated areas still occupied the anterior part of the posterior median columns, but no longer extended to the posterior commissure anteriorly, though they came correspondingly nearer the periphery of the cord. In the cervical region the degeneration occupied on either side a triangular area whose apex reached nearly to the posterior commissure, and whose base was formed by the periphery of the cord bounding the posterior median column. In other words, the degeneration was here more extensive than at other levels and occupied another part of the posterior median column. Above this level the degenerated areas seemed to occupy the entire posterior median column. Above the post-pyramidal nuclei the degeneration could not be traced.

The microscopical examination confirmed what has been said about the position of the secondary degenerations. It is interesting to note, however, that the degree of degenerative change in the area described was much less than might have been expected from the appearances; a considerable number of the fibres in these areas were normal, and the amount of interstitial tissue was slight. The appear-

ances made it highly probable that the tumor began in the gray matter of the left half of the cord.

There was nothing unusual about the structure of the tubercle itself. The appearances were strongly suggestive of its conglomerate character. Tubercle bacilli were found in small numbers in each section of the mass examined. They were confined almost exclusively to the giant cells which lay in the periphery of the growth external to its cheesy portion. Many of the giant-cells were without tubercle bacilli. Some of them contained one or two bacilli and a small number contained three or four. About the tubercle itself was a zone of white substance, containing normal nerve-fibres, but infiltrated in places with small spheroidal cells. In the left half of the cord the tubercle at one part comes to the periphery ; in other parts the zone of white substance is very narrow. There was no meningitis.

The points of special interest in this case are the rather rapid attainment by the spinal symptoms of a considerable degree of intensity, the great preponderance of the symptoms of tissue destruction over those of tissue irritation, of paralysis over spasm and pain, and the slight indications of intra-cranial disease.

CASE II.—Mary K., aged thirty-five, married ; housewife. Admitted to Roosevelt Hospital Feb. 27th, 1886, in the service of Dr. Watts. Family history negative. Patient began to menstruate at the age of fourteen, and was regular until seven months ago. She was married when twenty-three years of age and had six living children, one still-born one year ago, and a miscarriage seven months ago. Since this miscarriage the patient's health has been failing, her menstruation has been very irregular, and she has suffered much loss of blood from menorrhagia and metrorrhagia. She has also had a leucorrhœal discharge ever since the miscarriage, and has been troubled with pain in the back and limbs, and with headache. For some time before admission to the hospital she observed that the effort of lifting one of her children sometimes gave rise to nausea and vomiting.

Six weeks before admission the patient became conscious of some diminution in the power of the lower extrem-

ities. This loss of power increased gradually, and at the time of admission her legs had become so weak that she was unable to walk.

During the patient's stay in the hospital she frequently complained of pain in the legs, back and head. At times the headache was very severe. There was apparently an elevation of temperature most of the time.

It was noted at the time of admission that the knee-jerks were present, but an examination about twenty days afterward showed that there was bilateral loss of knee-jerk. At the same time it was noted that there were areas of diminished sensibility to pain, and areas of analgesia on the lower extremities, but the precise distribution of the sensory loss was not recorded.

The right leg was at this time completely paralyzed. The left was all but powerless. There was retention of urine from the time of admission.

On March 20th, when the patient had been in the hospital about three weeks, it was noted that there was a bilateral diminution of hearing.

On the day following a bed-sore began to form on the sacrum, and delirium set in. The delirium continued, rigidity of the muscles of the back of the neck appeared, and the head was rotated to the right. Internal strabismus soon appeared in the right eye, with dilatation of the pupil, and loss of light reflex of the same side.

On March 30th, a little more than one month after admission, the patient died.

The autopsy was made by Dr. Delafield. The lungs were found to be thickly studded with miliary tubercles. The spleen was rather small, and contained miliary tubercles. The cortical substance of the kidney was congested and was the seat of tubercles. The external os of the uterus was widely dilated. The wall of the uterus was thickened and its inner surface was congested and studded with very small miliary tubercles.

The walls of both fallopian tubes were uniformly thickened throughout their entire length with a greyish gelatinous material, which was nowhere cheesy. The stomach and intestines were normal. The pia-mater of the brain was congested and a little thickened and opaque, especially over the base, where there were also very small miliary tubercles. The lateral ventricles were distended with serum.

and there were very fine tubercle granules in the ependyma.

The pia of the spinal cord was infiltrated with turbid serum and thickly studded with miliary tubercles. Just below the brachial enlargement the cord was completely softened and broken down in about one inch of its vertical extent. In the lumbar enlargement of the cord was a spheroidal tubercular mass about three-eighths of an inch in diameter, with a cheesy centre, occupying chiefly the right half of the cord, but encroaching to some extent upon the opposite side. The mass was situated in the third lumbar segment of the cord. In the right half of the cord it replaced the grey substance and most of the anterior and lateral column at the level of its greatest diameter. The posterior column was intact in the greater part of its extent.

In the left side of the cord at this level the anterior horn was implicated, the white substance very little or not at all.

A microscopical examination showed the conglomerate character of the tubercular mass, and sections through the upper and lower end of the mass strongly suggested that the original deposition of tubercle was in the anterior horn of the right side. Tubercle bacilli were present in moderate numbers; the pia-mater was thickened and densely infiltrated with small spheroidal cells, and the periphery of the cord was similarly infiltrated, though there was here a tendency to the formation of distinct foci of inflammatory material. Many of the pial vessels were the seat of an advanced endarteritis. There was neither macroscopical nor microscopical evidence of secondary degenerations above or below the tumor.

It is interesting to note in this case, as in the one first recited, the relatively rapid development of symptoms indicative of an intra-spinal tumor, and the prominence of symptoms of tissue destruction as compared with those of an irritative character.

From the standpoint of spinal localization it is instructive to observe the relation which we must assume to exist in this case between the loss of knee-jerk and a destructive

process limited to the structures of the third lumbar segment of the cord.

CASE III.—This case is of interest chiefly from a pathological point of view. The clinical history is meagre. H. B., five years of age, gave a history of Pott's disease and of tubercular synovitis of the knee of some months duration. Both knee-joints were opened, and for nearly three months the patient did well. Then he developed the typical symptoms of tubercular meningitis, and died.

The autopsy revealed the existence of old and recent tuberculosis of the lungs. There was a well-marked kyphos in the lower part of the dorsal region, the bodies of two or three of the lower dorsal vertebræ being carious, and permitting the formation of a sharp angle externally. The diseased bodies of the vertebræ were in part replaced with cheesy matter and bone debris. Just above the lumbar enlargement of the cord the inflammatory products encroached upon the dura, but the cord was not visibly compressed. In the upper lumbar region of the cord itself was a small mass about the size of a pea, composed apparently of several grey tubercles. The brain showed the appearance of tubercular meningitis.

Microscopical examination of the cord showed the existence of a considerable degree of leptomeningitis, at the level of the bone disease and the grey tubercles just mentioned. This meningitis was most marked on the anterior surface of the cord, and was intense in the anterior median fissure. At the bottom of the fissure the inflamed pia was adherent to the substance of the cord. The grey commissure of the cord, especially its left half, and the anterior horn of the left side, were the seat of several tubercles, some of which had coalesced. The products of inflammation had nowhere become cheesy.

The precise limits of the tubercular infiltration of the grey substance could not be determined, as a segment of the left half of the cord had been injured. The process was apparently confined to the grey matter. The right side of the cord was intact. The central canal of the cord was dis-

placed to the left and its contour altered, but its lining epithelia were unchanged.

Tubercle bacilli were found in very small numbers in the giant cells.

There was no softening about the tubercular deposit and there were no secondary degenerations. The dura-mater at the level of the tubercle was altered in color in a circumscribed area of its inner surface near the anterior fissure of the cord. Examination showed the vessels of the dura at this spot to be enlarged and surrounded with accumulations of small spheroidal cells. The layers of the dura were also infiltrated with similar cells. The external surface of the dura was in contact with the inflammatory products resulting from the bone disease.

It is difficult to reach any conclusion except that there was in this instance a continuity of lesion between the bone disease and the tubercle of the cord.

It is instructive to examine the records of the cases of solitary tubercle of the spinal cord which have been hitherto published, in connection with the cases just recited, in order to determine what conclusions may legitimately be reached respecting the occurrence, clinical history and pathology of this disease.

Some of the cases published by the older authors are unreliable and must be partly or entirely discarded. In all, there are nine cases, of which the records are sufficiently detailed to enable us to form a picture of their essential features. These are the cases of Chvostck³ (two cases), Habershon,⁴ Eisenschütz,⁵ Williams,⁶ Kolitz,⁷ Obolonsky,⁸ Sachs,⁹ and Hayem.¹⁰

³ Med. Presse, 33-39, 1873.

⁴ Guy's Hospital Reports, 1872.

⁵ Tuberkel des Rückenmarks. Jahrb. f. Kinderheilk, 1870.

⁶ Deutsches Archiv f. klin. Med., xxv., 1880. Report of Autopsy by Schultze.

⁷ Ueber Rückenmarkstumoren in Kindesalt. Wien. med. Blatt., 41-43, 1885.

⁸ Ueber einen Fall von Rückenmarkstuberculose, etc. Zeit f. Heilk., Bd. IX, S. 400.

⁹ A Contribution to the Study of Tumor of the Spinal Cord. J. N. and M. Dis., 1886.

¹⁰ Obs. pour servir à l'histoire des tubercles de la Moelle épinière. Arch. de Phys. Norm., etc., 1873.

In addition to these cases there are a number of others, from each of which certain facts are available for statistical purposes. Such cases are those of Hellick,¹¹ Liouville,¹² (two cases), Serres, Dupurteau, Brichetlet, Tolling, Bichau, Gendreu, and Gull. Finally, Dr. Janeway of New York, and Dr. Thomas of Baltimore, have furnished me with some facts relating to the pathological findings in two cases of massive tubercle of the cord.

The study of twenty-four cases, in which the age is recorded, shows that solitary tubercle of the cord is a disease of the adolescent and early adult period of life, and of infancy, fifteen of these cases having occurred between the ages of 15 and 35, and five of them before the fifth year. Eight months is the earliest and forty-three years the latest period of occurrence noted. In eight of eleven cases in which the sex is given the patients were males. The facts do not warrant the expression of a positive opinion regarding the existence of a family history of phthisis, or of acquired syphilis, but in a large proportion of the cases after childhood neither of these conditions appear to have been present. An injury was noted as an etiological factor in only one case.

It would be superfluous to state that solitary tubercle of the cord gives rise to pronounced symptoms, were it not for the fact that some authors consider it not uncommon for this condition to give rise to no symptomatic manifestations. In one case, indeed, that of Schultze, a small tubercular mass occupied part of one lateral column of the cord and produced no symptoms during life. It is quite possible that in this instance slight symptoms would have been overlooked, but, granting that there were no symptoms whatever, this immunity might be explained by the circumstance that the patient died while the dimensions of the tumor were still very small, and by the fact that microscopical examination showed the nerve elements of the lateral column to have been displaced rather than destroyed.

¹¹ Considérations sur les lésions de la Moelle, etc. *Arch. Bolein. de Méd.*, No. 1, 1887.

¹² Nouveaux exemples de lésions tuberculeuses dans la Moelle épinière. *Arch. Gen. de Med.*, '75.

In all the other cases in which there were careful records, there were marked symptoms. In almost all these cases the earliest symptoms were pain and weakness of one extremity. Which of these symptoms is usually the earliest it is difficult to say, as the records are not sufficiently explicit. It is an important fact that both of these manifestations, together with the paræsthesia that often existed with them, were for a time either entirely or chiefly unilateral. Very soon the symptoms became bilateral. The loss of power became more marked, and in most cases ultimately became almost complete.

Anæsthesia was generally present. The pains continued and were associated with spasm. Irritative symptoms appear to have been present in every case in which the symptoms were carefully recorded. In a few instances they were intense. In the majority they were slight.

No statements can be made regarding the precise distribution either of the sensory or of the motor paralysis, as the data are lacking. Spasm does not appear to have been a conspicuous symptom. It was noted in more than half the cases. In one case it was very marked. In another it was present only on walking. In general the symptoms of tissue destruction have predominated over those of irritation, and this disproportion was very decided in cases I. and II. related in this paper.

The rapidity with which the symptoms have attained a considerable degree of intensity is a striking feature in eight of the nine cases in which the data enable a conclusion regarding this point to be drawn. In these eight cases the time that elapsed between the commencement of the symptoms and their attainment of marked intensity (considerable degree of paralysis, etc.), varied from three to eight weeks. No precise statement can be made regarding the time at which the symptoms reached their fullest development. Most of the cases appear to have run their course in about two months from the commencement of symptoms. In the case which forms an exception to this rule (Chvostek) the symptoms did not attain much intensity until seven months had passed.

In twenty-five cases of tubercular tumor of the cord only one tumor was described. In one case (Janeway) there were several, but the exact number was not noted.

It is certainly safe to assume that massive tubercle is solitary in a very large proportion of cases.

The records relative to the development of symptoms of tubercular disease in other parts of the body are unsatisfactory. They seem to justify the conclusion, however, that such symptoms existed in most of the cases. Usually the symptoms were those of pulmonary phthisis and occurred early in the course of the cord symptoms. They may be regarded, therefore, as a material aid in diagnosis.

Of sixteen cases in which the position of the tumor was noted, five were in the cervical region, seven in the dorsal, and three in the lumbar region. In the remaining case the tubercle was at the junction of the dorsal and lumbar regions. The cases are certainly too few to justify a precise conclusion regarding the relative frequency of massive tubercle in different parts of the cord, but they suggest that it occurs with about equal frequency in the lumbar and cervical cord, and that it is observed more often in the dorsal than in the cervical or lumbar region. It may be confidently asserted that the statement of Hayem and others that the lumbar enlargement is the favorite seat of massive tubercle is incorrect.

The description of previously published cases do not teach us much about the structural features of massive tubercle of the cord. The three cases here reported make it plain that in these cases the tubercular mass resulted from the coalescence of several tubercles, and it is probable that most, if not all cases of developed solitary tubercle are conglomerate in character.

In two of the three cases just mentioned the masses attained a considerable size and had cheesy centres. In the third case the tubercular deposit occupied an irregular and small area and had not undergone cheesy degeneration at any part. Tubercle bacilli were found in five cases (Obolonskey, Hellich, 3 cases here reported). They were present

in small numbers in four of these cases. In the remaining case (Hellich) they were abundant.

In the case of Sachs, and in the three cases which form the substance of this paper, the tubercular process occupied at first only one lateral half of the cord, and the unilateral character of the early symptoms in all the cases in which the early symptoms were noted, makes it more than probable that a one-sided growth of tubercle tissue is the rule.

Cases I., II. and III., of this paper indicate a beginning of the process in the gray matter, and in the case of Sachs the first deposition of tubercle tissue was in the vicinity of the posterior nerve-roots. In the case of Schultze, the beginning seems to have been in one lateral column.

In four cases secondary degeneration were found. In three of these there were both ascending and descending degenerations. In the fourth case there was a descending degeneration only. In ten cases no secondary degeneration was noted. Why these secondary processes were present in some instances and not in others, the facts at command do not enable us to decide. It may be surmised, however, that the ordinarily short course of the disease is at least a partial explanation of the absence of the secondary degeneration in so large a proportion of cases. In some of the cases (*e.g.*, that of Sachs) the duration of the process was longer than that required for secondary degeneration to occur, but it is to be remembered that the tracts which degenerate were probably not involved during this entire period. In case I., here described, the degenerative changes in the various tracts were only slightly advanced, a circumstance which indicates the importance of the time element.

Meningitis of the cord was noticed in several cases. It depended in most instances on the extension of meningitis from the cerebral meninges. Where the tumor grew so large as to reach the periphery of the cord a localized meningitis was sometimes set up. Softening about the tumor, sometimes of a myelitic character, was noted in more than half the cases. It was absent in the cases where the tumor

was small, and it is possible that its occurrence was related to the size of the tubercle.

There can be little doubt that solitary tubercle of the cord is generally secondary to tubercular disease in other parts of the body. Hayem, however, regards his case as one of primary disease of the cord. The lungs and intestines were the seat of tubercular disease in this case, and it appears most improbable that these parts should have been involved secondarily. It is possible that in some instances the tubercle bacilli may have lodged in the cord and in the other affected organs, at about the same time and in consequence of the same infection.

In eleven of twelve cases in which the histories give light on this point, tuberculosis of the lungs existed. In the remaining case there was tubercular meningitis. In none of the eleven cases just mentioned was the tubercular disease confined to the lungs and to the cord.

Tubercular meningitis existed in two of them. In two others there was massive tubercle of the brain, in several there was tubercular disease of the intestine, and in several tubercular disease of the kidney. In two cases there was tubercular disease of the uterus, and in one of them (Case II.) the uterine disease was probably primary. In one case (Case I.) there was extensive disease of the male genito-urinary tract, and here also the process was not improbably primary. In another case (Case III.) there was vertebral disease and tubercular arthritis.

It is more than likely that the lodgment of tubercle bacilli in the cord, on which we must assume the growth of the massive tubercle to depend, is effected through the circulation. Yet it is instructive to note that in rare instances the process extends to the cord by direct continuity of lesion, and probably through the medium of the lymphatics. This was certainly so in Case III. here reported.

It is believed that the clinical history of solitary tubercle of the cord can be more accurately written than heretofore with the help of the facts that have been recorded above. The uniformity of the symptoms in different cases, especially as regards their rapid course, the rapidity with

which the originally unilateral symptoms become bilateral, the comparative insignificance of irritative phenomena, and the frequency with which the signs of tubercular disease in other organs exist, are characters of solitary tubercle of the cord which may help in the distinction both from tumor of the membranes and from other varieties of tumor of the cord.

DISCUSSION.

Dr. SACHS said it was some years since his paper on this subject was written. At that time he had thought that the very slow development, the strictly unilateral symptoms and subsequent spread of the disease, involving both halves, would constitute a basis for diagnosis of tubercle of the cord. Now the point came up with regard to the possibility of distinguishing between extra and intra dural tumors.

THE MECHANISM OF INSANITY.

The "Journal of Insanity" for April, 1890, contains a paper by Edward Cowles, with the above title. First is considered the normal mechanism of mind and body, then reflex action. Following these there is an exposition of mental mechanism thus arrayed: consciousness; apperception; attention; sense-perception; memory, imagination; conception, reason, judgment; feelings, emotions, conscience; willing and acting; and subconsciousness.

THE EFFECT OF ANTIPYRIN ON THE SOLUBILITY OF CAFFEINE.

The "Therapeutic Gazette," April 15, 1890, calls attention to the fact that antipyrin possesses the property of increasing the solubility of quinine salts and likewise that of caffeine. Added to caffeine in equal parts, antipyrin renders the drug perfectly soluble in cold water. (See "Repertoire de pharmacie," Feb. 10, 1890.) With warm water, it is claimed that it is possible to dissolve seven and one-half grains of caffeine in one hundred and sixty minims of distilled water, by the addition of twelve grains of antipyrin, and that the solution so produced is permanently limpid. This increased solubility of caffeine favors its subcutaneous administration, since through its aid fifteen grains of caffeine may be dissolved in three ounces of water. Thus the use of the drug in neuralgia and migraine becomes greatly facilitated by the addition of antipyrin, and the probability of cure increased.

ON GOLD CHLORIDE AS A STAINING AGENT FOR NERVE TISSUES.¹

By HENRY S. UPSON, M.D.,

Attending Physician to Lakeside and City Hospitals, Cleveland, Ohio ; Neurologist to the
Dispensary of the Western Reserve University.

THE object of the present paper is a consideration of the chemical relations of gold chloride, as it is used in staining nerve fibres and ganglion cells. This is in effect a continuation of two articles on the same subject, already published in the JOURNAL OF NERVOUS AND MENTAL DISEASE ; in order to give as complete a view of the subject as is possible, a partial repetition will be necessary.

The Freud method, which has been in use for some years, is as follows : The section, cut from tissue hardened in Müller's fluid, comes first into a 1% solution of gold chloride, where it remains for two hours. It is then brought successively into a 20% solution of potassium hydrate, and a 10% solution of potassium iodide ; in the latter solution in a few moments it assumes a reddish tint. If successful the nerve fibres and ganglion cells are sharply stained. Or occasionally, for some unknown reason, the myeline sheaths alone are stained, presenting an appearance much like that of a section stained according to the Weigert method. This stain has been little used, as it is said to be quite unreliable.

Now, the formation of the red or purple oxide of gold from the chloride is a chemical process, and as such follows fixed laws. It is the result of the combined or consecutive action of oxidizing and reducing substances on the chloride ; so that it is probable *a priori* that if we are sufficiently well acquainted with the substances used in the reaction, and the way in which they act, so that we can

¹ Read at annual meeting of the American Neurological Association, June 4, 1890, in Philadelphia.

make the conditions in all cases practically identical, we shall be able to obtain uniform results.

The objects of the investigations summed up in the present paper are two-fold ; namely, to find chemical agents which shall determine a formation of gold oxide in the tissues in such a way as sharply to differentiate them ; second, to determine the mode of action of these agents, so as to make the methods of their application reliable.

The method which, in a previous article, I have designated as No. 1, is briefly as follows :

METHOD NO. 1.

The section cut from tissue hardened in Müller's fluid (or much better in a simple 2% solution of potassium bichromate) is brought for a time varying from ten minutes to several hours, into a 1% solution of gold chloride ; then for a moment into water, and into a 10% solution of potassium hydrate, where it remains for 1-2 minute ; then, after a moment in water, into a reducing fluid, consisting of

Sulphurous acid, 5 cc.
Tr. iodi 3%, gtt. x.
Liq. ferri. chlor., gtt. i-iii.

This method depends for its success, in the first place on the presence of a chrome salt in the tissues. Hardening in Müller's fluid is essentially a reduction of the potassium bichromate by the fresh tissue, with an appropriation by the tissue of chromium, probably in the form of an oxide.

That the chrome salt is deposited in such a way as to differentiate the nerve elements, is seen by a glance at the cut surface of a bit of nerve tissue hardened in potassium bichromate. The tracts of nerve fibres which show on the surface may be followed with great accuracy ; to make this differentiation available for microscopical purposes, it is only necessary to replace the chromium by another metal, whose oxide has a sufficiently intense color to be plainly visible when under the microscope.

It is this oxide of chromium, which is replaced by gold chloride when the section is brought into the gold solution,

and if it has been soaked out of the tissue in alcohol or water, no stain can be obtained. This necessitates a staining of the section within a reasonable time after the removal of the tissues from the bichromate solution.

In the second place, the stain is dependent on the presence of extraneous matters in the gold solution. Pure gold chloride, dissolved in distilled water and carefully neutralized, stains little if at all. It is not too much to say, that every section which has been successfully stained by the older methods, has been so by virtue of hydrochloric acid or some other impurity accidentally present in the gold solution. The commercial chloride of gold almost always contains hydrochloric acid, often also chloride of copper, and a solution which is in use soon contains varying quantities of chrome salts, acquired from the sections which have been brought into it.

Much better results may be obtained by adding substances designedly to the gold solution, and these substances should be, in general, either acids or metallic salts, or both.

The acid which is most useful alone is hydrochloric acid ; those which may be used with the metallic salts are hydrochloric, nitric, sulphuric and chromic acids.

The metallic salts which are most useful are ammonium vanadate, potassium permanganate, sodium stannate, and others.

In this method, No. 1, the potassium hydrate may be used in solution alone, but if it is so used the stain is often too diffuse. In this case substances may be added to differentiate the color, giving a sharper stain to the nerve elements proper, and leaving the glia tissue little stained, if at all. Ferricyanide of potassium is the most powerful agent of this class. The ferrocyanide acts in the same way, but more weakly.

There is another principle which it is necessary to bear in mind in all operations with metallic salts ; it is this : chemical agents which have just been set free are very active, and ready to enter into new combinations. In order that they should be vigorous in their action, the reducing

fluid should be freshly made for each set of sections to be stained, and the potassium ferricyanide should have been recently added to the hydrate solution. The gold solution, on the other hand, may be used for an indefinitely long time.

It is evident that with all of these means at command for varying the process, the number of different combinations which may be employed is endless. It will suffice to give one of them in detail.

The section to be stained is brought from water into a 1% solution of gold chloride, to which has been added 2 or 3% of hydrochloric acid. Here it remains for two hours, more or less ; after being washed superficially in water it is then brought for $\frac{1}{2}$ to a minute into 10 cc. of a 10% solution of potassium hydrate, to which has just been added a piece of ferricyanide of potassium as large as a pin-head ; it is better to add the latter salt in the solid form, and to ensure rapidity of solution it may be kept powdered and a quantity about corresponding to the above dissolved in the potash solution.

The section after this may be brought for a few moments into a 10% sol. of pot. hydrate to remove the excess of ferricyanide, and then, after washing a moment in water, comes into the reducing fluid made as follows :

Sulphurous acid, 5 cc.
Tr. iodi, 3%, gtt. x.
Liq. ferri. chloridi, gtt. i.

The section at once assumes a bright, purplish red color, and in a moment or two should be removed to water, and mounted in balsam by the usual manipulations.

The quality of the stain as affected by variations in the process will be considered later.

The above may be taken as a type of the process in Method No. 1.

In order to have well in hand the manipulations which may advantageously be used, it is well to begin with a series of sections cut from a bit of well-hardened spinal cord, and, after staining as above, to try the effect of adding dif-

ferent acids and metals alone and in combination, to different portions of the gold solution, and testing the staining powers of each. A considerable variety of results will be obtained in this way, and the methods thus learned may be adapted at will to the state and quantity of chrome salt in any given set of sections.

METHOD NO. 2.

This differs from Method No. 1 mainly in the fact that the reducing fluid contains a salt of tin. This change, however, necessitates a few variations in the other steps of the process.

To the gold solution may be added a metallic salt, or a salt and an acid.

To the hydrate solution should be added, in place of the ferricyanide, a trace of ammonium vanadate, and potassium permanganate; the latter substance has the same differentiating power that is possessed by the ferricyanide in the preceding method.

On the whole this method gives better results than does the preceding one; it should be varied to suit differences in the hardening, since it is obvious that a section thoroughly impregnated with chrome salt from a long stay in the bichromate solution, may endure, and indeed require a more vigorous differentiating or decolorizing action than would a tissue not so well hardened.

The method adapted to a moderately hardened tissue is as follows:

To 5 cc. of a 1% solution of chloride of gold, add one or two drops of a 1% solution of potassium permanganate, and one drop of nitric acid; this solution is permanent, and at any rate should not be used until two days after it is made.

The section, after two hours in this gold solution, is brought for a moment into water, and then into 10 cc. of a 10% potassium hydrate solution, to which has just been added a trace of ammonium vanadate and one or two drops of a 1% solution of potassium permanganate. Here it re-

mains for $\frac{1}{2}$ to a minute, is brought for a moment into water, and then into the reducing fluid, made as follows :

Tin solution,	gtt. xv.
Water,	3 cc.
Iron solution,	gtt. ii.
Sulphurous acid,	3 cc.

What has been called tin solution above, is made by adding 3% tincture of iodide to a small amount of pure protochloride of tin, until in the solution the reddish color of the iodine begins to be apparent.

The iron solution is a saturated aqueous solution of phosphate of iron, the soluble scale salt being employed.

The section almost at once assumes a deep purple color in the reducing fluid, and is removed to water and mounted in balsam as before.

When the sulphurous acid is added to the reducing fluid, an active chemical change takes place, and a heavy precipitate falls ; it is while this reaction is going on that the solution is most efficacious, and the section should be brought into it at that time.

Sulphurous acid is a gas, which occurs in commerce dissolved in water ; it must be kept in tightly glass-stoppered bottles, and should be obtained as fresh as possible.

With tissues which have been long and thoroughly hardened, a gold solution may be employed containing, instead of the above, ten drops of a saturated solution of ammonium vanadate, and two drops of hydrochloric acid, to 5 cc. of a 1% gold chloride solution.

QUALITY OF THE STAIN.

This varies considerably, according to the care with which the various steps of the process, and especially the hardening, are carried out. In the comparatively unimportant matter of the exact shade of color taken by the nerve elements, it is not always possible to predict the result, as a slightly greater or less intensity of reducing action may change the result from reddish to bluish purple, and *vice versa*. In general it may be said that hydrochloric acid in

the gold solution gives a reddish color in the section, and that metals without acids in the gold solution give a more bluish shade.

Without doubt gross errors in the staining process proper are capable of preventing a useful result from being obtained, but if with moderate care a good result cannot be obtained in test sections by either method, the trouble is invariably in the hardening of the tissue.

In case of such failure to obtain good results, the possibility should first be considered, of the chrome salt having been soaked out from the tissues, as in that case the task of obtaining a good stain is quite a hopeless one. Next the hydrate solution should receive attention, as too large a quantity of ferricyanide or other substance added to it is quite capable of decolorizing not only glia, but nerve tissue as well. Attention to these two points will, with a moderately well hardened tissue, be sufficient to insure success.

As with all other methods, the best results are only obtained by careful handling of thoroughly well hardened tissues.

HARDENING.

The object of the hardening process should be clearly borne in mind.

The Weigert method depends for its success on the presence of a chrome salt acting as a mordant in the myeline sheaths; tissues hardened for a long time in Müller's fluid often appear dark green, almost black, in the white matter, a light gray color in the gray matter; these tissues stain well by the Weigert method; they give with gold chloride a distinct myeline sheath, but weak ganglion cell and axis cylinder stain.

The object to be attained in hardening tissues for the Golgi stain is a comparatively rapid permeation of the lymph spaces; the appropriation of the chrome salt by the tissue itself appears to be not so important.

Quite fresh tissues hardened for a long time in a weak solution of bichromate of potash, present no very marked difference in color between the white and gray matter; but in the gray matter may be seen, on the cut surface of a bit

of spinal cord, the groups of ganglion cells, and bundles of nerve fibres radiating out into the white matter. Tissues hardened in this way give with gold chloride a clear ganglion cell and axis cylinder stain, but probably stain badly by the Weigert method. They stain well with carmine and nigrosine.

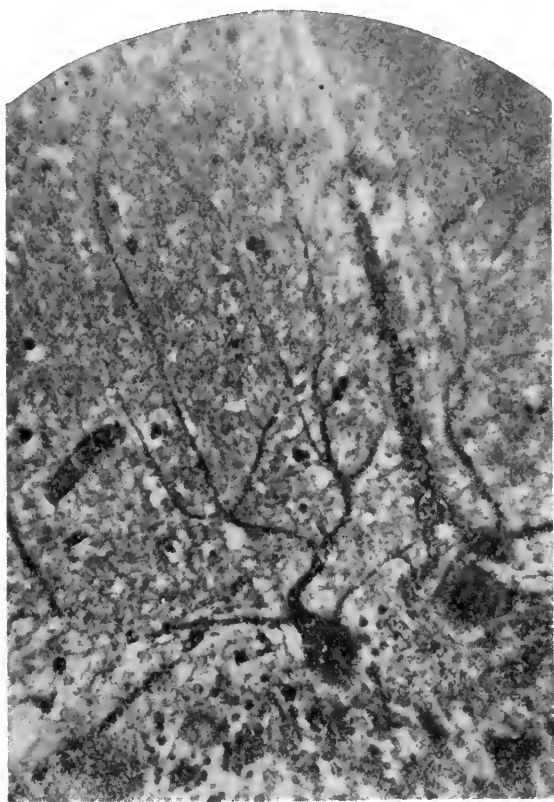


FIG. 2

Fresh tissues should be brought for a week into a 1%, then into a 2% solution of potassium bichromate, and should remain in the latter for from three to six months or longer; the hardening in this way proceeds more slowly than when sodium sulphate is present, but the chrome salts penetrate much better, and act more uniformly throughout the tissues.

The tissues should be cut into rather small pieces, and the solution changed often.

That only well-hardened tissues can be successfully stained with chloride of gold is by no means so disadvantageous as at first sight appears. Even gross tissue changes are often obscured, and what is worse simulated, by *post-mortem* change both before and after the commencement of the hardening process, and this no matter what stain is

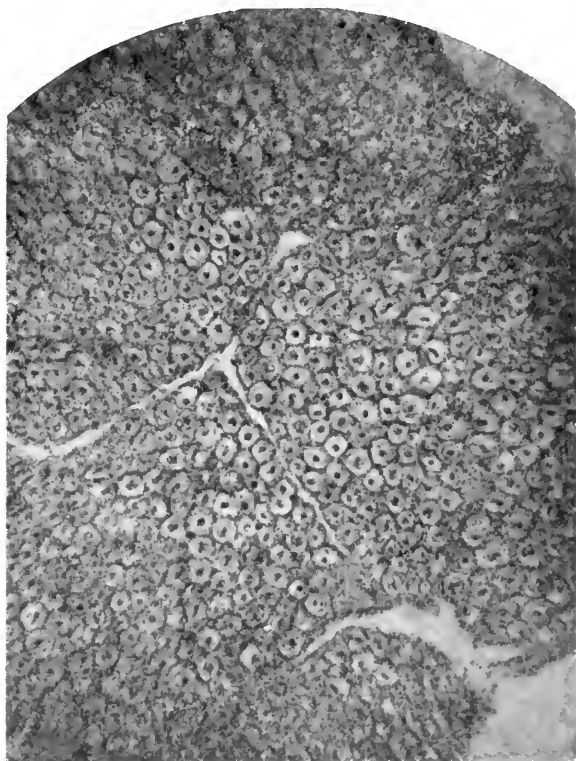


FIG. 1

used ; the finer nerve fibres are not very unlike the surrounding glia in chemical constitution ; it is probable that many forms of disease are due to a finely molecular or chemical change in the relations of these parts, and any attempt to investigate such conditions must be undertaken in tissues which have been preserved as nearly as possible in the relations which obtained during life.

The merit of gold chloride is not that it is easily availa-

ble in tissue no matter how badly hardened, but that with care nerve elements may be more sharply differentiated than with carmine or nigrosine, and I believe with as uniformly good results.

The accompanying photo-micrographs are from untouched negatives taken from sections stained by the above methods. They were taken with a Zeiss microscope, objective DD, eye-piece No. 3, and a Schmidt detective camera



FIG. 3

without lens. The plate was about six inches from the eye-piece. An oil lamp furnished the light, which was passed through an Abbé illuminating apparatus. The plate used was a Seed 26 extra rapid, and the exposure two to five minutes. The half-tone reproductions are not so clear as is a bromide contact or albumen print.

The first micrograph is from a section of the normal human cerebellum, and shows several cells of Purkinje, with

their processes extending into the gray matter. The second is a cross section of a human nerve trunk, taken from the spinal canal. The third is from the spinal cord of an ox, and shows a large motor ganglion cell in the anterior horn of gray matter, surrounded by other cells and nerve fibres. The tissues were all normal, hardened in Muller's fluid, and stained, No. 2 by the first method, the others by the second method.

SOME SEDATIVES FOR INSANE PATIENTS.

Dr. O. Dornblüth (Kreuzberg), *Therapeut. Monatshefte*, 1889, Aug. —; *Neurolog. Centralblatt*, No. 4, 1890). Dornblüth experimented with hyoscine hydrobromate (E. Schering), and obtained the same results as with Merck's preparation. The remedy was mostly given internally in soup, coffee and wine, in doses of 2 neg. two to three times daily. As a secondary action vertigo, a tired feeling and dryness of the throat were observed; illusions were only seen once and in a nervous woman who mentally was normal. After subcutaneous employment of the drug quite frequently a loss of control of the extremities, dryness of the throat, etc., appeared. The action of subcutaneous injections, as a rule, is observed after three to ten minutes, while that after the remedy is internally administered in one-half to one hour, if the patient be kept quiet after taking the drug. In ten per cent. of the cases an initial stage of excitement, sometimes of two to three hours, would precede. The remedy was especially valuable in periodic mania and continuous maniacal states; also in epileptics, hallucinatory patients, paralytics, and patients suffering from dementia, it acted sedatively. He also experimented with codeinum purum and phosphoricum, subcutaneously, in doses of 0.025 to 0.05; internally, 0.02 to 0.08. In quiet melancholic patients, doses of 0.06 to 0.08, two to three times daily, acted still better than the "course" of opium already tried. In maniacal conditions of excitement codein acted not so well as hyoscine. It acted very well in anxious restlessness, præcordial sensations and sleeplessness. The patient, according to Fischer-Kreuzlingen, does not form a codeine habit. The phosphate is only to be used subcutaneously, as codeinum purum is soluble with difficulty. No secondary or disagreeable after-action appeared. In a slight degree of excitement he began with 0.02, and with grave excitement 0.04.

TWO CASES OF FRIEDREICH'S DISEASE.*

BY CHAS. HENRY BROWN, M.D., OF NEW YORK.

I ESPECIALLY desire in this paper to report two cases, which I consider clinical types of that interesting form of hereditary degeneration of the spinal cord designated as Friedreich's disease.

I select these two, because they differ in many points clinically, though they both serve to illustrate some interesting points for consideration as to the pathological nature and extent of the disease.

Miss H., age twenty-nine.

Family History.—Both parents living. The father is of the neurotic type. Emotional; delicate. Discouraged easily and often has broken down under business cares.

The mother is also of the emotional neurotic type and suffers from migraine. She has a brother in the asylum. A sister with narcomania, and another brother of very eccentric habits and mental peculiarities. There were six children.

The oldest is the patient, Miss H.

The next is a man of twenty-seven, very much undersized, and of delicate physique.

The next is a man of twenty-five, in good health.

The fourth is a girl of nineteen, very much undersized, and in appearance and actions undeveloped; suffers from migraine. Has menstruated from thirteenth year of her age. Menstruation has been irregular. At times profuse, at others scanty. At times appearing two and three weeks apart; again not appearing for two months. Suffers from profuse nose bleeds. Has a small undeveloped uterus.

The fifth child, a boy, was delicate from childhood. Very nervous and slightly choreic—in fact, lived with diffi-

* Presented to the American Neurological Association at its annual meeting held in Philadelphia, June, 1890.

culty to the age of sixteen, and died with a particularly short attack of pernicious anæmia.

The sixth child is a robust boy—developing into a apparently strong manhood.

Personal History.—The early history of the patient was uneventful to near the age of puberty. She had then attacks of supposed malaria. She menstruated at thirteen. At this time she consulted the late Dr. Knight for being very round-shouldered and ungraceful in her movements. He diagnosed double lateral curvature. The patient wore a steel corset for one year, night and day, and another year during the day only. The curvature, however, increased, and she grew more round-shouldered and ungraceful.

In August, 1876, age fifteen, she had an attack of supposed rheumatism; no swelling; only pain in the joints of the extremities.

In spring of 1878, there was so much headache, backache, and erratic pains in the extremities that the girl was obliged to leave school. At this period ungracefulness led to frequent falls, and she was still supposed by physicians to be suffering from rheumatism. The patient had a number of attacks of pain in head, back and extremities, each lasting from two to three weeks and each followed by more than the usual ungraceful gait up to 1882, when a more than usual severe attack of headache and backache left her unable to walk without assistance or stand without swaying, and if it was attempted with eyes closed she was liable to fall. She had suffered since puberty, up this date, with attacks of cramps in the calves of her legs, mostly occurring at night, very soon after she fell asleep.

Patient talked badly and had a disagreeable eruption on knees, elbows and knuckles. Elevated papules, capped by scales. This eruption lasted two years and slowly disappeared. She consulted Dr. L. A. Sayre in 1882, who treated her double lateral curvature by extension and placed her in a plaster jacket. The patient, however, grew rapidly ataxic. In 1883 she suffered from profuse and frequent menstruation, being obliged to arise as often as twelve times at night. She was unable to perform fire

coördinating movements with the hands. Her facial expression was unmanageable. The muscular movements were overdone. For instance, a smile was a grimace; an attempt to wink caused a grotesque facial spasm.

The slurring and rapidity, with omission of labials, made speech perplexing.

Nystagmus was an early symptom, but the exact time it was first noticed is not remembered. It was at first most constantly present when the patient was about sixteen years of age. The patient has suffered more or less constantly with erratic pain in head, back and extremities.

I first saw patient April 7, 1884. She is still under my professional care.

Examination.—A handsome girl; blonde, slight and small of stature; well-nourished; intellectually bright; no mental derangement. Patient had a marked ataxic gait, unable to walk or stand alone. Sitting, patient exhibited marked ataxia of body muscle, ataxia of upper extremities, face and speech muscles. All muscular movements awkward and overdone. Tactile sense good. With æthesiometer variable and slight anæsthesia noted. Poor weight sense. Heat and cold were easily distinguished. Abolition of the deep and superficial reflexes. Electrical examination negative. Ophthalmoscopic examination negative. No pupillary symptoms. The double lateral rotary curvature of the spine noted.

Summary to Date.—The patient has had variable attacks of headache and backache. Vaso-motor troubles noted in irregular action of heart at times. Flushings of face, extremely cold hands and feet, with chilblains in winter. Polyuria at times. Attacks of pulsations in groins and vulva, with heat and swelling of the latter, lasting days. Ataxia increased slowly, especially of the lower extremities. Muscular strength fair. The patient has preferred to remain in bed most of the time of late. There is no muscular weakness sufficient to absolutely confirm a paresis supervening.

This is undoubtedly a case of early systematic degener-

ation of the posterior columns, extending to medulla and various vaso-motor centres.

The prominent clinical features, tabulated as follows, are to be seen in typical cases of Friedreich's disease :

1. The age of development.
2. A general ataxia.
3. No fixed anæsthesia.
4. Nystagmus.
5. Ataxic speech.
6. Abolition of reflexes.
7. Curvature of spine.
8. Vaso-motor symptoms.

This group of symptoms form the most general ones noted in this class of cases.

The atypical or anomalous symptoms are :

1. Initial pain.
2. Spasms.
3. No apparent essential paralysis.

The initiatory attacks of pain, however, lacked the character of true lancinating pain of tabes. They were more constant, and confined to head, back and joints; and this character of pain has been often recorded in cases of Friedreich's disease. Griffith found these initial pains in 22 of the 143 reported cases he had analyzed.

The cases reported by Mussa, Botkin and Friedreich, all suffered from initial headache. Bradbury reports a case with pain and weakness of the back.

One of Carres most typical cases suffered with severe initial pain in legs and feet. Pain concomitant with the disease, and after it is established is not so uncommon.

Forty-five cases tabulated by Griffith had neuralgic pains of various nature. The attacks of pains in my case were more frequent after the disease was noted and well established. Pain is merely anomalous symptom, inasmuch as it is not a precursor of Friedreich's disease with that severity as noted in tabes.

What causes the pain in tabes has been a question for much discussion. Meningitis, degeneration of posterior roots, or peripheral neuritis. This is uncertain as yet,

though degeneration of posterior roots is conceded the chief cause.

Why the pains of ataxia should exceed those of Friedreichs' disease is of interest. Déjerine, in the discussion on the nature of the sclerosis of Friedreichs' disease before the Société de Biologie, March 7, 1880, claimed that the lesser difference in degree and character of the pain, in the disease under consideration than in tabes, is not due to unequal or less alteration in the posterior roots, for in the cases under his observations the posterior roots were as much diseased as in tabes. But the fact of a difference in the character of the degeneration might explain why we are more apt to have severer pain with tabes than in Friedreich's disease.

In the former we have the degeneration closely allied to an inflammatory process, while in the latter we have a disease undoubtedly depending upon an evolutionary process.

It is easy to understand that, in the breaking down of young and most delicate tissue less resistance is met with and, consequently, less response in functional disturbances. In this case of mine pain was a factor, and the question naturally suggests itself, was it not due to a more active disturbance in the breaking down of older and therefore stronger and more resisting nerve tissue, and to deduce the inference that had the degeneration occurred at an infantile period than at puberty and adolescence, that the process of dissolution would not have been accompanied by pain, or at least not much.

It is natural to surmise that at puberty more active and irregular disorders of functions are apt to occur and all symptoms to assume a more active type.

One of the interesting features of this case is regular occurrence of muscular spasms at night. This is not of unfrequent occurrence in the reported cases; but I will leave its discussion till I take up some analogous features with the next case.

Muscular weakness would naturally be expected to be present more or less in a case of advanced ataxia.

In the reported cases of Friedreich's disease this symptom has not been specially spoken of in many cases. The paralysis was not considered an important feature, and the inability to walk was not in all cases related to any degree of paresis present. Griffith gives 56 cases as pronounced ones exhibiting more or less muscular paralysis. Again, many cases reported were in young cases where the disease was not advanced. Whether the inability to walk in time will result in secondary complications and degenerations of the lateral tract is a question.

The case I report is certainly far advanced in her ataxia, and has for years been unable to walk much or has used her limbs to any extent, yet muscular weakness is not pronounced and a true paresis is not present, and whether the case develops essential paralysis certainly is a question.

The following case has been described by Dr. Dana,¹ in an article entitled "Combined Sclerosis of the Spinal Cord. His description was complete, but differs slightly in a few particulars from that obtained by myself:

Jane D., aged twenty-eight; single; no occupation.

Family History.—The grandmother (father's side) had epilepsy. The father, when a young man, drank, and suffered from frequent "nervous spells" with vertigo, and always complained of indigestion, and has doctored ever since he could remember. The mother had been physically a weak woman many years before this child was born. She has had three young children die in convulsions at the ages respectively of sixteen, eleven, and eight months. She has three grown daughters, all of whom were very nervous as children. One, now married, is reported to be in fair health. Another, aged twenty-one, has often complained of having strange feelings and thoughts with uncontrollable morbid impulses to run and shout; suffers from attacks of migraine, and is hysterical from any slight physical or mental strain.

¹ Medical Record, July 2, 1887.

Personal History.—As long as the mother can remember the patient was a nervous child. She had no physical endurance, tired easily; she had frequent attacks of mental confusion, imagining she was in other places, with complete loss of personal identity or those of friends and relatives that might be around her. These attacks evidently were epileptoid.

From description patient had attacks of psychical blindness. Troubles with vision were frequent, and, as she grew toward the age of puberty, she became more disinclined to exert herself. She menstruated at fourteen, and has been regular, though the flow always was very scanty. No history of genito-urinary troubles.

She has lived a quiet, uneventful life, without vicious habits. As her disease progressed, she has shown less and less hysterical and epileptoid symptoms. The symptoms were prominently apparent when about sixteen. She was obliged to look where she was going or she would be apt to fall; and she walked badly, and stumbles and falls were frequent. This was followed in a year by numbness and tinglings, with some weakness, and the numbness extended to the hands, more in the right than the left.

In 1884 these symptoms were constant, with considerable weakness in the legs. From this time symptoms became rapidly worse, with coldness of extremities, slight puffiness of feet, formications, and slight delay in act of micturition. Her feet felt like rubber balls on the floor; she staggered when she walked, and the heels struck the floor first.

At this time inco-ordination was the marked symptom, though she was weak and limbs were slightly stiff, with occasional cramps in muscles; but she never had acute pain. She could not use her hands for delicate co-ordinating work, such as sewing: the needle was not felt and held securely without the aid of vision, and great labor was necessary to make the stitches even.

1885 and 1886.—She grew worse, and became unable to walk. Stiffness with cramps was present a great part of the time, but diminished the latter part of 1886, and con-

tinued to do so up to my first seeing the case in the early part of 1889.

Examination.—She was well nourished; complained of not being able to walk or to use her hands freely. By assistance could take a few steps; at times able to go about the room with her sister's aid. Limbs not stiff; ankle-clonus elicited as well as tendo-reflex; more marked on re-enforcement.

Slight anæsthesia of both lower extremities, with slight hyperæsthesia of both plantar surfaces. Heat sense good; weight and space sense poor.

In the upper extremities muscular weakness marked; more noticeable on right side; right arm feels also more numb. No reflexes obtainable; slight anæsthesia: heat sense good; weight sense and space sense poor.

The extremities always feel very cold, and are abnormally so. Electrical examination negative; ophthalmoscopic examination negative; pupillary reflexes normal; no nystagmus; no curvature of spine; speech is slow, but not ataxic. Patient is eccentric, stubborn, and slovenly.

January 1, 1890.—Patient suffered from a sudden attack of diplopia (right sixth nerve paralysis), with drawing of muscles of left side of the face and paresis of the right side, and vomiting. This was a probable central hemorrhage, and will not be further considered in the discussion, except to say it is now hardly discernible.

This case of early progressive degeneration of the cord presented first marked ataxic symptoms, followed by ataxic paraplegic condition, with spastic symptoms for a time.

The anomalous symptoms are the quickly developing paresis with spastic symptoms. The presence of the reflexes and ankle-clonus are difficult to explain, unless we accept Westphal's explanation in like cases: that knee-jerk will not be abolished unless the lumbar enlargement be involved. And Griffith, in his summary, concludes that genuine cases may occur in which the reflex are slight, normal, or even exaggerated.

In twelve cases reported the reflexes were present; in

six exaggerated. Spasm of muscles occurred in twenty-one cases, and marked ankle-clonus in two cases.

There is no marked spastic condition in any of the muscles of the lower extremities in this case now. This was a symptom occurring in the more active stage of the disease.

Strumpell speaks of the hereditary nature and family type in spastic ataxia, and one he reports² which resembles mine in many particulars. Dr. Dana speaks in his article of the importance of heredity as a factor.

In spite of the quickly developing paresis and the presence of the reflexes with spasm, I think this case must be classed in the category of the hereditary degenerations. It is probable that the degenerations occurring in the various tracts in both these cases were due to evolution, the cord in part being predestined to early destruction.

In many cases of Friedreich's disease it is conceded that it is not necessary to have an exact type of the disease in a parent or ancestor or even a correlation with a neurosis or neuropathic taint. It has been found, however, that the 143 cases reported by Griffith occurred in 77 families.

There has been an endeavor on the part of investigators to seek for the family type, and more attention has been paid to the reporting of these than to the isolated cases. This undoubtedly leads many to look upon the isolated cases as not typical ones of Friedreich's disease, and especially if they in any way present anomalous symptoms.

Many just such cases as those I report, which differ markedly from the acute degenerations of the adult, and yet not directly traceable to heredity, point strongly to anomalies of development of probably ectodermic origin.

To lay too much stress upon the imperfect nervous mechanism of ancestor or parent as the cause in these cases is not practically wise, and is apt to draw attention away from the consideration of other unknown (perhaps physical) forces at play in the vital structure of the blastoderm.

It is of great scientific value to study, as near as possible,

² Arch. f. Psych., vol. xvii., No. 1, case iii.

the embryonic origin of the tissue itself, which undergoes a retrograde change, and the character of the pathological evolution.

The importance of histogenesis of tissue to pathological study is becoming more apparent. Blastodermic derivation of tissue is being carefully investigated in new ways, as from the side of morbid anatomy. Formad³ demonstrates that the derivation of the tissue determines the variety of new growths in that tissue and their character of development, so that prognosis and diagnosis can readily be made. The character of the pathological evolution of tissue is toward that of the early derivative tissue.

Chaslin demonstrated that in epilepsy there exist cortical gliomata in the form of fibrillary tissue analogous to that of the neuroglia, without any vascular lesion or sclerosis.

Déjerine and Letulle, in a paper on the nature of the sclerosis in Friedreich's disease,⁴ discuss the histological differences between this form of degeneration and that of tabes or other sclerosis. In Letulle's case the degenerative changes were of the neuroglial character.

Tangled fibres of various lengths and every possible arrangement. Nothing of note was to be found in connective-tissue change in blood-vessels, except slight hyaline degeneration of the lymphatic sheath of the capillaries. Prolongation of pia mater was normal and intact; this, however, was chiefly located in the posterior columns: more positive evidences of connective-tissue sclerosis of a vascular origin being found in the lateral columns and other tracts.

Gliomatous masses were seen as well about the central canal. This gliomatous histological nature of sclerosis in Friedreich's disease unmistakably points to anomalies in development and the evolution toward derivative neuroglial tissue.

The evolution in Friedreich's disease follows more or less a regular order, with undoubtedly a tendency to implicate the entire cord. It insidiously selects the posterior columns first, creeping on and spreading out into other portions

³ University Med. Mag., May, 1890.

⁴ Le Médecine Moderne, March, 1890.

slowly. There is undoubtedly a more active stage, which completes more quickly and for all time the gross extent of the lesion, and the disease again halts and the degeneration progresses more slowly. In my first case remissions and exacerbations were marked and extended over several years.

The implication of all parts of the cord in the degenerative process has lately been observed by Blocq and Marinescu.⁵ In abstract it is as follows:

Considerable diminution in volume of the entire cord, which is abnormally small in any given area. The entire columns of Goll are affected throughout—the column of Burdach irregularly—as far as the decussation of the pyramids.

The crossed pyramidal tracts are entirely changed, the lesion decreasing from below upward as far as the decussation. The cerebellar tracts are affected from the lower dorsal region.

Gower's tract is not abnormal within this region. Lissauer's zone is the seat of disease in lower and middle lumbar region. The marginal external tract is unaffected. The columns of Clark are profoundly changed throughout their entire length. Histological lesions occur even when there is no appearance of gross abnormality.

Fewer large healthy fibres than there should be are present; and there is an alteration in their disposition. There were changes in the funiculi gracilis, the cerebellar tracts, and the funiculus cuneatus of the medulla. Besides these various degenerations, there was vascular dilatation of lacunæ form existing throughout the changed tissue. *In toto* an entire implication of the cord and a destructive degeneration of a great portion.

The stereotyped clinical phenomenon observed in Friedreich's disease is that of ataxia with more or less paralysis, and these complicated irregularly by myopathies, deformities, vaso-motor derangements, and even cerebral difficulties.

⁵ Le Progrès Médical, March 8, 1890.

The variations in the ataxia relatively to the degree and extent of the paralysis, and more or less prominence at times of the complications, leads one to classify cases often as types of ataxic paraplegia, spastic paraplegia with some ataxia, or again perhaps as a case of imbecility with ataxic paraplegic complications.

To classify the variations of Friedreich's disease into types will not aid in the better understanding of the true nature of the disease, and will not be satisfactory, as no two cases are exact in various clinical phenomena. Anomalies in the clinical picturing are to be expected, but it will not be difficult to adjust these when we recognize the nature of the pathological process and its rather extensive irregular distribution without fixed limitation to the posterior columns or even posterior columns and lateral tracts.

THE PARALYSES OF LARYNGEAL MUSCLES DURING THE COURSE OF TABES DORSALIS.

Dr. R. Dreyfuss (*Virchow's Archiv*, 1890, Bd. 120, p. 154), after a historical review, gives the results of his investigations, made in Mendel's Polyclinic. There were twenty-two patients in all. He found in two patients paralysis of the posterior crico-arytenoid muscle in the first stage of development. In the other patients laryngoscopic examination did not reveal any motor disturbances which could be said to be due to tabes; in all the cases there was a total lack of change in sensation. The writer then gives the description of three other cases of tabic paralysis of the vocal cords.

He objects to the use of the term "real ataxia" of the vocal cords in tabes, proposed by Krause, for one can only speak of ataxia of a group of muscles; but on inspiration only one muscle in the larynx, the posterior crico-arytenoid, comes into action. The cases of phonetic ataxia of tabes described up to now only present this disturbance of co-ordination as transient; hence this symptom is not persistent. He does not by any means consider tabic laryngeal affections as frequent as Krause (thirty-three per cent.) and Marina (fifty per cent.).

CLINICAL EVIDENCES OF BORDERLAND INSANITY.*

By IRVING C. ROSSE, A.M., M.D., F.R.G.S., WASHINGTON, D. C.,

Professor of Nervous Diseases, Georgetown University.

THE name that I have taken to include the forms of mental degeneration about to be considered may not be the most appropriate one from a strict psychiatric point of view. However, when we take into account the difficulty in studying the relation between sympathetic instinct and the cerebro-spinal experiences, as well as the vague line that is supposed to separate reflex action and volition, it is hardly worth while to confine one's mental perception to names, or to definitions and classifications that are purely arbitrary and comparative.

Everybody knows of positive and negative electricity; of black and white; of health and disease; of high spirits and mental depression, but our knowledge of the imperceptible difference in their intermediate conditions is extremely limited. Analagous to this is the study of the phenomena of a class of persons standing in the twilight of right reason and despair—a vast army whose units, consisting of individuals with minds trembling in the balance between reason and madness, are not so sane as to be able to control themselves, nor yet so insane as to require restraint or seclusion.

For the scientific mind the clinical observation of cases of this kind carries with it a sort of fascination; the analysis of the facts relatively thereto seems to be the order of the day, and is a subject worthy of occupying attention and exercising our sagacity. Their study being of comparatively recent date, contemporary psychiatry has created

* Read at the annual meeting of the American Neurological Association in Philadelphia, June, 1890.

from them a special class, the so-called pseudo-momaniacs, who not only show certain well determined psychic disturbances, but at the same time are conscious of the unusual phenomena taking place in their nerve centres, and are capable of discussing and even describing their intellectual and moral derangements.

Aside from clinical consideration patients thus affected are of forensic importance, since the study of their malady touches some special medico-legal points particularly delicate and obscure. The solution of such interlocutory questions as may arise from the latter point of view being rather juridical than medical, I shall take for granted the pathognomonic character of the malady in question, and without further generalization shall mention a few typical illustrations selected from the experience of my own practice as a neurologist.

For obvious reasons the cases are shorn of many personal details, yet the omission is not such as to interfere with the material facts.

The first case that I shall mention is that of a young man, a clerk, in whom there was an aberration of the genesc instinct. He consulted me, upon the advice of Dr. N. S. Lincoln, for a morbid impulse that had troubled him for some time. The impulse was homicidal, and manifested itself in an almost uncontrollable desire to kill some member of his family, though for what reason he could not tell, as he had no idea of committing a crime, and was well aware of the difference between right and wrong. The trouble was unknown to his family, although the impulse to kill his father had come so irresistibly while sitting at breakfast, that in order not to yield to it, he suddenly quit the room and afterwards came to my office in a state of great agitation.

I found at first but little in this young man's antecedent's to account for his neuropathic state. He had no marked peculiarity of look, speech or action, and a long series of questions, put with a view to lead up to a mental weak point or delusion, failed to elicit any other symptom. The great bodily functions were apparently normal, with the

exception of that of the generative organs. Among other things, he informed me that for more than a year he had almost nightly visited a young woman whom he kissed and fondled in a lascivious way, but without accomplishing the act of sexual intercourse.

In this case it required but little diagnostic and pathological acumen to trace the genetic connection between the brain and testicles, and to direct the treatment accordingly.

When I last saw this patient he was fresh-colored, well nourished, and otherwise apparently well.

Subsequently to this I was consulted by a Washington lawyer with a large practice as patent attorney. This man was overworked, his system was below par; he had insomnia; absence of the patellar tendon reflex; *clavus-hystericus* on the right side of his head, and was myopic. He complained of pain in his right eye; of inability to use his mind for more than ten minutes at a time, and labored under the notion that he was becoming deranged and would soon wind up in an asylum. The thought of his faculties being compromised he had kept more or less in a latent state by the influence of his will, so that the trouble was unknown to any one except his medical adviser.

I had the patient carefully examined by a competent ophthalmologist, both in Washington and New York, with a view to find out the cerebroscopic indications furnished by the eye, as well as to correct any ocular defect that might account for a part of the symptoms. The treatment that followed being only partly successful, I persuaded the patient to quit work and go to Europe for a short trip. He did this with much benefit; but some months after his return the symptoms came back with renewed vigor.

I advised another long sea voyage on board a sailing vessel from Baltimore to Rio Janeiro and back. This trip was followed by highly satisfactory results to the patient, who, saved from the stigma of an asylum, regained his mental health, has since married, and is now entirely well.

In February, 1884, I was consulted by a prominent business man of Washington, of middle age, who had

suffered from obstinate insomnia and its train of evil consequences for eleven years, and during that time had contracted the bromide habit. He had hypochondria, morbid fear, impotency, and a suicidal impulse. In addition to treatment by local physicians, he had been under eminent neurologists in Philadelphia, New York, London and Paris, with but indifferent results. His atavistic antecedents were not entirely satisfactory, but his venereal history was fairly good; he had stopped the use of tobacco, and his habits were temperate, though he was not a teetotaler. The patient complained of general languor and debility; of a clawing pain within the head; of inability to concentrate his thoughts; of loss of will power, and a fear of insanity. He said that he had taken the bromides of potassium and sodium in enormous doses since 1873. For two years, from 1874 to 1876, he took daily 135 grains without missing a day. The quantity was gradually decreased in 1878 to 80 grains a day, and to 50 grains in 1883, during a visit to the "Healing Springs" of Virginia. He subsequently took 90 grains daily. The patient was cachectic; but there were no sensory, motor, or mental symptoms indicative of any gross organic lesion. Examination with the differential calorimeter, however, revealed the existence of a considerable degree of cerebral hyperæmia.

Under treatment my patient made but indifferent progress towards improvement. I had nearly exhausted my efforts to break up the bromide habit, when Dr. Wm. A. Hammond informed me that he had cured several cases of this habit by prescribing a solution of chloride of sodium, the dose of which is gradually increased, the bromide, at the same time, being correspondingly decreased until a few minims is reached, when the patient breaks off the habit on being told that he is taking nothing but common table-salt. This plan did well for several weeks, but during my summer absence in Europe the patient accidentally learned from his druggist what he was taking, and immediately discontinued the prescription. On my return in autumn, in a fit of sheer desperation, I directed the patient to eat large quantities of grapes, the object of which was explained to him, and within a week

I had the surprise and satisfaction to see a salutary change. The psychical depression was much less, and the craving for the bromides was nearly gone. The habit is now broken up, the patient sleeps fairly, has regained his virility, is rid of the morbid impulse, and is bodily and mentally reconstructed.¹

A patient, in a state of fear and having the idea of persecution, came to me in November, 1887, in the person of a middle-aged German with a wild, uneasy, restless eye, and a suspicious manner.

He had conceived the notion that his instability was the result of his parents being much under the influence of wine at the time he was begotten; that he was sexually out of order; that he was being persecuted generally; and that he would soon be hopelessly insane.

Like many of these patients, he showed a morbid propensity to write letters, one of which details the subjective symptoms; so I will give them in his own words:

"The most foul and degrading remarks are made about me in the office I am employed in by at least one person—aside from everything else, such is the case, and simply for the purpose of annoying me. I know this. I would not mind such remarks at all, but the effect of the uttered words on my imagination is actually a terrible one; the oddest combinations are formed, and I am almost completely subdued by them in my actions, motions, &c. All this could to some degree be avoided if I was not so morbidly sensitive; still the effect is there.

"A couple of years ago I noticed numbness in my feet. This occurred while I was walking in the street; it struck me as very peculiar. I had similar feelings on several occasions at that time, but they have since disappeared.

"Last year and the previous one I noticed that I frequently and painfully bit my tongue while eating, in an almost awkward manner. I ascribed this as a paralytic symptom. Such occurrences I have not since experienced.

"After meals I feel that my stomach has to perform considerable work. I get a hot and heavy feeling in my

¹ See *The Medical Record*, New York, October 10, 1885, p. 417.

head, which lasts for a considerable time, and which is accompanied, especially in the morning, with considerable nervousness and excitement.

"While talking with persons in the office, it seems as if I was constantly ashamed of something, which is comparatively and partly true.

"If I have to stand up when a person speaks to me, I have the desire to sit down or to take hold of something or rest my hands and elbows on something. Very often my head begins to swim; I have a feeling of falling forward and to cast my eyes down.

"I have had moments, not two months ago, when I became indescribably alarmed, as if being on the verge of losing my mind and becoming violent. This has left me since I stopped drinking coffee.

"As a fact, I have gone through all kinds of fixed ideas; I thought I had mastered them all; but now of a sudden my worst apprehensions have been excelled by persons actually expressing aloud my innermost thoughts.

"Aside from all inherited defects of mind and thought, my present trouble seems to me to be the morbid state of my sexual, mental, and physical condition. And, as remarked before, any allusion to my person in that respect makes me act almost like an hypnotic.

"Respectfully yours,
"_____."

Any one familiar with such cases as the foregoing knows how few are the objective symptoms and how unsatisfactory is their treatment. Such patients, never remaining long with one physician, ultimately become the prey of quacks and charlatans, or may become hopelessly insane.

My latest knowledge of this man is that he had fallen into the hands of an itinerant quack, who mulcted him of several hundred dollars and took his notes for several hundred more.

In September, 1888, a business man, of a highly neurotic family, several members of which I had treated for nervous affections, came to me on account of his distracted mental condition. Two of his family had already died insane, and he imagined that he was going the same way. His alcoholic, venereal, and nicotinic habits were of the worst, and had left their stamp in his physiognomy, and I may even

say in his speech and action. It would be too long a story to relate the numerous symptoms, subjective and objective, most of which were clearly traceable to constitutional syphilis.

I placed this patient on the twentieth of a grain of biniodide of mercury three times a day, and, being a man of means, I directed him to go to Atlantic City, to take hot baths of sea-water, and lead a quiet, sensible out-door life. Under this regimen, which the patient observed scrupulously, the cerebral symptoms soon faded away. He regained his former health, but a year subsequently relapsed into dipsomania.

Among functional intellectual troubles that may arise from morbid fear, not the least curious are those affecting the feelings or emotions in the more recently determined conditions known as moral hypochondria, agoraphobia, and the peculiar morbid condition that syphilis may sometimes give rise to. The last named I witnessed in a case of syphiliphobia in a young naval officer, who came to me on September 10, 1889. He had contracted syphilis some years previously, and had apparently been treated successfully, but having married, and his wife being pregnant, he developed a well-marked case of mental invalidism without the appearance of any distinctive syphilitic lesion. After the manner of most of these cases, he had consulted eminent medical men in various cities, both at home and abroad, who had equally failed with myself to give satisfaction. Remaining under observation but a short time, he quit me for another physician, and I have since lost sight of him.

A type in which the morbid trouble is more distinct and better defined is to be found in agoraphobia, or, as it is sometimes called, kenophobia. For the last two years I have had under treatment a well-marked case that is subject to exacerbations. Two other patients that I have cannot look up to the dome of the Capitol or the summit of the Washington Monument without being seized with agoraphobic symptoms. The three cases occurred in foreigners,

men of intelligence and education, who had kept the symptoms concealed from every one as long as possible, for fear of being thought insane.

I wish I could dwell longer on this interesting form of psychic insufficiency; but having written up the subject in Buck's "Reference Handbook of the Medical Sciences," I must refer those interested to that article.

The next case to which I shall call attention is that of a priest of twenty-eight years, who was dyspeptic, emaciated, and hypochondriacal. When a boy of seventeen he had cerebro-spinal meningitis, from which he convalesced slowly and had become deaf. What troubled him most, however, was the fact that he had lost faith in the tenets of the religion in which he had been educated. For the last two years he said that remorse had seized him, and being shrouded in spiritual darkness he fell into a lethargy of despair, cursing the sun each morning, and hoping every day would be his last.

This case convinced me that the gastro-intestinal condition and religion are somewhat interdependent. I directed the treatment accordingly, and soon had the satisfaction to see both mental and physical improvement, after which the patient left for his home in one of the Southern States.

Another case in which religion entered as an element occurred in a boy of seventeen, whom I was called in consultation to see at a private sanitarium near Baltimore in February, 1886. Having become much preoccupied with religious matters, he developed into a case of hysterocatalepsy, and was in a pitiable condition when I saw him, being emaciated, motionless, and speechless. He also had the waxen flexibility of limbs peculiar to the cataleptic state, and the least touch about the face caused such strong contraction of the muscles that it was necessary to give his food and medicine with a pump after forcing open the mouth with a gag in which was a round hole.

The patient made rapid improvement under treatment, and in a few weeks returned to his home near Washington,

where he became strong and well under a regimen in which severe muscular exercise and suppression of the emotions were the prominent factors.

Unhappily, this young man's religious instinct was again perverted in the summer of 1889. On a chilly day in November he came home about dinner-time, refusing to speak or to eat, and showed symptoms of his former malady. These rapidly developed into acute mania, from which the patient died in the sanitarium where I first saw him.

A curious case that has puzzled me much is that of a young man of twenty-one who had partial paranoia with pseudo-aphasic ideas. He was referred to me by Dr. Godding, of the Government Asylum for the Insane, in May, 1889. This man was employed in a newspaper-office, where he attended daily to his duties without having incurred suspicion as to his mental state. Being something of a student, and having lately read numerous works on psychology, he conceived the notion that he was suffering from a perversion of the ego; that he was unable to perform an act of judgment; that he had lost all his faculties save that of memory; and that all his movements were simply automatic and in no way influenced by will-power.

Having exhausted the gamut usual in examining such cases, I was unable to discern any physical reason that might account for the mental symptoms. The patient assured me that his habits were good, and he did not have that furtive, uneasy, restless look peculiar to victims of secret vice. The more noticeable features of this case were the perversity in using the pronoun "it" in reference to vague symptoms which he could not or would not explain; the ingenious manner in which he answered my questions; and the specious arguments he used in discussing his case with me—this, too, in view of the alleged fact of his inability to construct a mental process, and his belief that he was suffering from aboulomania.

An occasional aloetic aperient with a course of ergot and bromide was followed by a breaking up of the fixed idea regarding his automatism and memory; and a month

later I succeeded in convincing him of his ability to perform an act of judgment to the extent of constructing a syllogism, though he could not "distribute the middle third." He also recognized the fact that he had *willed* to come and see me and to do many other acts, but he could not break up the troublesome habit of introspection, and vagueness in regard to the "it" was still as pronounced as before. In fact, there was a persistent misuse of the pronoun when he should have used a noun. In this respect he seemed to be suffering from a kind of aphasia, which showed itself in an inability to use nouns. Two months subsequently the patient still showed this phenomenon of the impairment of the function of expression. His articulate speech did not permit of its being outwardly construed or interpreted. Yet at this time the patient acknowledged that he saw an improvement in his judgment, and that he had got rid of some of the functional derangements of which he formerly complained. The latest feature of this case is the fancied loss of all emotional control, although to the observer this is no more apparent than the fabulous wealth of many general pareitics.

That the brain cortex was impaired in the next observation I think will be readily admitted. It is that of a middle-aged man who was dyspeptic, used tobacco and alcohol to excess, and suffered from obstinate insomnia. Among other peculiarities he informed me that he was a "crack-walker," and would not for any consideration knowingly step on a crack of the flag-stones when walking in the street. He had also developed the curious phenomenon known as mirror-writing. This functional agraphic condition was unaccompanied by paralysis. He wrote with the right hand, in a reverse way from right to left, anything that I could dictate. The act was done with the rapidity of ordinary writing, and the chirography was so good that when held up to a mirror it was easily read. This man is intelligent and educated, converses rationally about the peculiarities of his case, and believes that he will die insane. He is at present in business in New York.

In connection with the foregoing case it may be worth recalling Leonardo da Vinci's reversed manuscript in the Milan Library, which many neurologists think is the result of an attack of right hemiplegia, and that he had become incapable of writing otherwise.

Akin to this nervous instability of the "crack-walker" is the morbid fear arising from the neglect of something very trivial. Most of us know of the mild manifestation of morbid mental energy related of Dr. Johnson's left foot, and of his peculiarity in touching certain lamp-posts with his walking-stick. Something like this I have lately seen well illustrated in the case of a wealthy retired merchant, an American of more than average intelligence, who, while sojourning abroad at Nice, had a morbid fear of going to bed unless he had walked every day to the end of the Promenade, some distance from his hotel, and there touched with his walking-stick a certain cactus. On several occasions he went to bed without going through this little daily performance; but he had invariably to get up, dress himself, and repeat the episode before his fears had sufficiently subsided to enable him to sleep. On a subsequent visit to Washington the same alteration of the nervous level occurred. He was obliged each night to walk from Willard's Hotel to the Capitol and there touch the curbstone with his stick, or else sleep was impossible.

The mental disturbance so common about the period of the menopause may be classed in the same category as the cases just mentioned. Among other patients of this class that I have had under observation for the last year is a woman of more than usual intelligence and character. Like many of her sex, she had an unhappy love-affair in her early days, and had never married.

She was neurasthenic, had insomnia, facial paralysis, lagophthalmia of one eye, and complained of tingling and numbness in the lower extremities. Her mind had taken a decidedly erotic turn. She talked freely and with great prolixity of her sexual starvation, of the mysteries of life,

of copulating with a Newfoundland dog, and of other obscene subjects. In addition to this moral obliquity, the power of attention and of memory was weakened.

Most of the bodily symptoms in this case have yielded to treatment, but mentally the patient is still hanging around the borderland. Her latest freak, shown in a propensity to write, has brought to me a correspondence that may be looked upon as a psychic autobiography. Every week or so she mails to me a letter with a special delivery-stamp. The letters are well written as far as regards grammar and choice of expression, but the ideas therein are vague and show a weakening of the logical inhibitions. One of these letters tells me of a miracle that had happened to her: that she was walking in the street, feeling so depressed as to be nearly insane with wretchedness, when suddenly she had a strange experience; the operation of natural law had stopped in her being; a beatific feeling came over her, followed by the most perfect peace and quiet, and a voice spoke to her words of consolation and good advice.

Her last letter to me was not so much wanting in the mal-assimilation of its mental components as it was in relevancy. So far as it relates to anything in my mind it is totally incomprehensible. It reads as follows:

" Now listen to a true friend, and do not imagine that any one knows, for they do not. I detected a woman watching some one's house one Sunday evening when I was starting out for church.

" That is enough for a woman who has seen such things duplicated. It is one of those cases in which a true friend can take no part except with damaging results to all and no benefit to any.

" Don't let a band of evil-doers break your friend up, and don't let him do that which men so frequently do (but women rarely).

" Running away from the effects of some one else's evil-doing is neither courageous nor manly. Don't let your friend neglect his business hours or impress any one that something troubles him to the neglect of business. There is always some honorable way out of all complications in

which an innocent party is a victim. Your friend will come out all right. Don't let him get the blues.

"The more hydra-headed they show themselves the better for him.

"Your friend,

"———."

In December, 1889, I was called to see a young unmarried man, a lawyer from an adjoining State, who had come to Washington for rest and recuperation from a late mental illness. From his brother and himself I learned that he had lately been engaged in an important lawsuit, the prosecution of which had incurred overwork, anxiety, and sleeplessness. Having finished his case in court, he was waiting for the train on the platform of a railway-station, where he was the subject of a visual hallucination. To his great surprise his law-partner, whom he had just left, stood beside another man at the end of the platform, and, on approaching to shake hands with him, the apparition disappeared. Turning away and walking toward the other end of the platform, the same thing occurred, to his utter terror. He subsequently suffered from obstinate insomnia and the monomaniacal ideas of paranoia, which showed itself in the belief that various persons were trying to prove him dishonest in his profession and to persecute him generally. Although conscious of the delusion, he was unable to reason himself out of it.

There was nothing that I could elicit from this patient's hereditary or atavistic antecedents to denote the existence of a *vesania* or a *neurosis*. His alcoholic and venereal history was good; but he was a great smoker, and had had a domestic trouble, the details of which it would be irrelevant to mention.

Salient features of this case were impaired general health, dilated pupils, hebetude, and a slow staccato way of talking after the manner of speech noticed in *katatonia*.

After a few weeks' treatment directed to restoring the general health and relieving insomnia, the symptoms gradually faded away; the patient recovered, and is now well and attends to the duties of his profession.

In studying the prodromal symptoms of psychical impairment, one seldom meets with a more curious condition than filth-dread or mania of contamination, the so-called mysophobia or rupophobia.

A case that I have come to me on May 7th. His father visited my office before I had seen the patient, and, in telling what he had noticed as peculiar in his son, unwittingly gave a very good clinical history of the affection. This boy had developed the utmost horror of becoming contaminated. The fear of defilement extended to nearly all his surroundings, both at home and in the street, and it needed constant assurances, with the exercise of much parental firmness, to relieve his mind. Aside from touching ordinary objects, after which he subjected his hands to innumerable washings, the fear of the pollution extended more particularly to greenbacks and to using the water-closet, which he did with great reluctance, after covering the seat with clean towels, and would in no circumstance touch the handle that flushed the bowl. He had a morbid fear of having his hair touched, a dread of shaking hands, of coming in contact or running against any one in the street, and an unclean or badly dressed person caused such loathing that he would cross to the opposite side.

On examination I could find no appreciable somatic cause for the condition in question. There was perfect integrity of perceptive aptitude, acoustic, tactile, and visual stimuli, and the patient talked intelligently of what he considered exaggerated peculiarities that he was unable to control by his will. At my suggestion patient has lately crossed the Atlantic in a sailing ship, and at last accounts was doing well.

Whether this morbid fear of contact be only an anomaly of the sensibility and instinct or an undeveloped paranoia, I am unable to say. The psychopathic symptoms mentioned in this case do not belong to the full-blown variety; they occupy a sort of neutral ground that is neither Spain nor Gibraltar.

I shall not attempt to formulate any general conclusions from the neuropathic states and mental anomalies that I

have endeavored to portray in the foregoing recital. But I may say that the study of cases showing rudimentary indications of insanity is of more value, from an educational point of view, than that of a fully-developed case. The early recognition of the functional derangements that precede the outset of confirmed insanity is often a matter of great difficulty. Such cases rarely come within the experience of asylum physicians, and being subjected to neglect in the incipient stage of the malady, they eventually go to make up the larger proportion of incurable lunatics.

I trust I may be pardoned for saying again that such cases are of practical importance from a juridical point of view. Who, for instance, would not question the criminal responsibility or the civil capacity of nearly all the persons just mentioned, after knowing their clinical history? In the case of a crime or a misdemeanor, who can discern clearly whether one of these has yielded to an unhealthy and irresistible incitement, and not to the suggestions of his interests or his passions? Or, in other words, whether the act would be a morbid phenomenon, and not a passional phenomenon? A question might also arise in the matter of contracts, or as to the validity of wills of such persons.

Since lawyers are dependent upon medical knowledge for enlightenment in such equivocal cases, they resolve themselves into a simple question of medical diagnosis; and the only way out of the embarrassment which will lead to a correct judgment is the rigid application of general diagnostic means and the ordinary proceedings of clinical investigation.

TWO CASES OF TUMOR OF THE CEREBELLUM.¹

BY J. ARTHUR BOOTH, M.D., NEW YORK,

Assistant Physician Manhattan Eye and Ear Hospital.

IN calling attention to these cases, I do not do so with the idea of announcing anything novel or of bringing forward something new as to the localization or the pathology of the disease, but only because I believe that all such cases ought to be recorded, especially when autopsies have been made and the diagnoses verified.

The first case was under my observation from the commencement of the patient's illness and closely followed to the end. The second case I saw only two days before death.

CASE I.—Sarah D——, aged ten years. Seen in consultation with Dr. Z. S. Webb on February 19, 1888.

Parents healthy; no family history of cancer or tuberculous disease. The child had not had any previous serious illness. When three years old she fell down a flight of steps, striking her head upon the stone walk. The fall could not have been a serious one, as the parents did not recall the accident until after her death, though questioned carefully before as to traumatism.

About November 1, 1887, the parents noticed that she was losing flesh and becoming very pale.

Ten days later she came in one afternoon, complaining of chilliness and headache, followed by vomiting without any nausea. She slept well that night and seemed perfectly well the next day. On November 15th and 20th she had similar attacks of headache and vomiting. Each attack seemed more severe, and the recovery not so complete. She was also becoming very irritable—did not wish to play, talk, or be disturbed. A little later the father thought she carried her head as though the neck was a little stiff.

¹ Presented to the American Neurological Association at its annual meeting, held in Philadelphia, June 4, 1890.

From January 15, 1888, the child commenced complaining of pain in the back part of the head and neck, principally in the morning before getting up, but always wanted to be dressed and go down-stairs to her meals. She now spent most of the time in her mother's lap. The parents had not noticed any staggering in her gait, nor did she at any time complain of dizziness.

Examination (February 19, 1888).—Child dressed and sitting in mother's lap; head bound with cold compresses. She is dull, listless, complains of severe head pain, and does not like to be disturbed; is too weak and miserable to get down and walk. Speech normal; tongue straight; no tremors; vision seems normal to finger-test; pupils dilated; no reaction to light. There is a slight paresis of right external rectus; no nystagmus; no changes in the fundus. Grasp of hands fair; no paresis of face or limbs; knee-jerks absent; no anæsthesia. There is marked sensitiveness to touch all over head, and especially on back of neck, just below occiput. The posterior cervical glands are quite large. Temperature in axilla $101\frac{3}{4}^{\circ}$; pulse 100, irregular.

Diagnosis.—"Tubercular meningitis."

Tumor of the cerebellum was considered; but the absence of optic-nerve changes and the presence of an elevated temperature with an irregular pulse led me to give the former opinion. Besides, many of the facts in the previous history of the case I was not able to obtain until later, especially as to the vomiting.

Patient was ordered ten grains of the iodide of potassium in milk every four hours, this dose to be increased five grains each day.

February 24th.—General conditions worse. Pains in head still present; vision same; pupils not so large and react to light. Paresis of external rectus has disappeared. Temperature 100° ; pulse 120; respiration 18. Ophthalmoscope showed beginning optic neuritis; vessels small and indistinct. Is taking to-day thirty-five grains of potassium iodide every three hours in milk. Vomited after second dose. To take grains twenty as before.

March 5th.—First well-marked convulsion occurred,

which was followed by many others, five or six during the day. These consisted of tonic spasm of limbs, drawing up of right side of lip and ala of nose; left eye wide open and right eye tightly closed. Low moans and sometimes a loud scream would accompany these attacks. Father states that on two occasions only was consciousness entirely lost.

The symptoms certainly point to an intracranial growth, and are probably due to a cerebellar lesion.

March 9th.—Very much prostrated; vomits the iodide, which had been reduced to ten grains every three hours. Temperature 100°; pulse 118, thready and irregular. Stimulants given as necessary, and patient ordered syr. ferri iodide, twenty drops three times a day; also inunctions of cod-liver oil.

March 10th to 20th.—There is a slight improvement. Vomiting ceased; appetite is good; patient swallows her food easily. Convulsive seizures less frequent and less severe. She passes her urine involuntarily, and the act is generally accompanied by a seizure, as before described.

Examination of Urine.—1018, alkaline. Traces of albumen; no sugar or casts. The abdomen, which had been sunken in, rounded out; and the whole body seemed to flesh up once more. She is still very irritable; repeats the words of others and any sounds she hears outside; also uses strong language, quite often saying "devil," and even worse. The use of these expressions quite surprise and shock the parents, who cannot imagine where the child could have heard them.

March 25th.—The only new symptom is dimness of vision; does not distinguish objects beyond four feet. Ophthalmoscopic examination shows advanced atrophy of both optic nerves.

July 1st.—Child is entirely blind; lies most of the time in a semi-comatose state; occasionally attacks of petitemal. There is now paralysis of right side of face, partial paraplegia and paresis of left arm. The following bulbar symptoms have also appeared: Dullness, impaired articulation, difficult deglutition, and polyuria

August 1st.—Emaciated to an extreme degree; cannot

swallow; is fed with a tube. Left arm and both legs contracted and rigid. There has been a gradual enlargement of head, and there is now some separation of coronal and sagittal sutures. Slight exophthalmus present. Patient died on August 17, 1888.

Autopsy on August 18th, nine hours after death, the head only being examined.

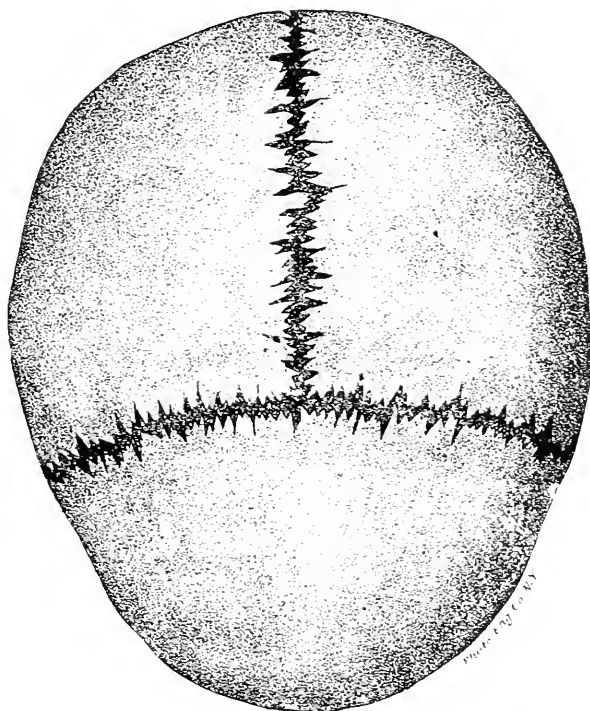


FIG I.—Degree of Separation of the Sutures.

The whole head was very much enlarged, the frontal and parietal bones being very thin and separated at the sutures to a marked degree (Fig. I.). The dura mater was very thick and distended. On puncturing this, a large amount of clear fluid gushed out. The convolutions were flattened and whole brain was pale, flabby, and softened. The lateral ventricles were very much dilated and contained a large amount

of fluid. The medulla was flattened, compressed, and softened.

Base of Brain.—Olfactory bulbs normal. Optic nerves small. Other nerves not examined.

Cerebellum (Fig. II.).—Placed directly between the lateral lobes of the cerebellum is a large nodular growth, three inches long, one and one-quarter inches wide, and one and

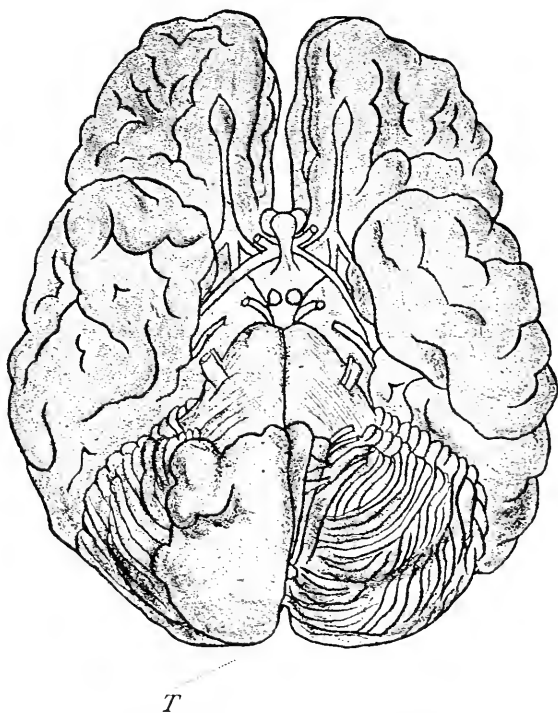


FIG. II.—Base of Brain. *T* points to Tumor in Situ.

one-quarter inches in its vertical diameter. It extends into and is attached to the right lobe of the cerebellum, occupying quite an extensive hollowed-out space in this lobe (Fig. III.). The growth also extended into the fourth ventricle, pushed the medulla to the left, and pressed on the right crus of the cerebellum.

Dr. Frank Ferguson makes the following report of the microscopical appearances of the tumor:

"The tumor is nodular in outline, in places cystic. Examination shows a large number of spindle-cells, medium in size, imbedded in an abundant granular and fibrillated stroma with a rich vascular supply. The walls, being composed of embryonic tissue, are quite thick, and give the tumor the appearance of angio-sarcoma."

CASE II.—James S., aged thirty-five years; married; bookkeeper. Seen in consultation with Dr. M. Tygert, Rutherford, New Jersey, on March 20, 1890.

Has always been much occupied by his business, giving but little time to recreation, often not returning until late at night and then continuing his work for several hours. His general health was always good, with the exception of an occasional headache, until last August. At that time

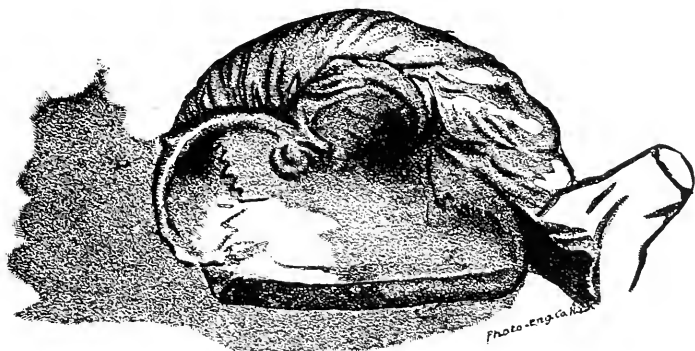


FIG. III.—Depression of Right Lobe of the Cerebellum, into which the Tumor fitted.

he commenced to complain of pain and discomfort in the back part of the head and neck; was more irritable and could not apply himself as formerly without causing fatigue and increasing the distress in his head. The pain seemed more severe on rising. Later in the same month he had a fainting attack, as he was about to go up-stairs. His wife states that there were no convulsive movements, no nausea or vomiting. His extremities were cold and lips blue. He recovered from this attack in a few minutes; was assisted to bed, passed a comfortable night and seemed well the following day. No staggering in gait noticed at this time.

During the summer and fall the pain in occiput became more constant and troublesome, though not preventing his attending to business, which necessitated a daily ride of

twenty miles on the cars. There gradually developed an uncertainty in his gait, a sort of swaying and a tendency to fall to the left or forward. He commenced to lose flesh and strength. The head pain became more acute, and finally, four weeks ago, he was obliged to take to his bed. Occasionally he would get up and go to the bathroom adjoining, and it was noticed that he would stagger and would have to keep hold of the furniture, etc., to prevent himself from falling.

For the last three weeks there has been present almost constant hiccough, with collections of mucus in throat. Lately he has had some difficulty in swallowing, and at times his speech has not been distinct. Ten days ago he complained of food collecting between his cheek and sides of teeth on left side. Memory has been good. No trouble with vision. No dyspepsia. Bladder functions normal. Bowels regular.

There was no specific history. Has had two children, both healthy. Wife one miscarriage. No family history of consumption or cancer. Mother still living. Father died of paralysis.

Examination.—Patient in bed, lying on left side, body bent forward and downward. Answers questions intelligently. Occasionally a word would be pronounced rather thick and indistinct. Voice fairly strong. Lies motionless and does not like to be disturbed. Complains of severe pain over occiput and back of neck. Over the second and third cervical vertebræ there is a large denuded surface, the result of cantharides which had been applied for the relief of pain. Glands of neck quite prominent. There is no tenderness to pressure on any part of head. Muscular system not well developed. Muscles of extremities flabby. Tongue deviates slightly to the right. Not tremulous. No anæsthesia of face. Slight loss of sensation over tips of fingers of left hand. There is a marked paresis of lower facial muscles of left side, also of left arm and hand. Raises hand to face slowly, with difficulty, and in doing this shows pronounced ataxia. No paralysis of the ocular muscles. Left pupil larger than right, reaction to light not as good. Opens and closes both eyes equally well. Nystagmus in a lateral direction is present to a marked degree.

Ophthalmoscope examination reveals neuro-retinitis in both eyes. Grasp, R. 20—19; L. 5, 4. Muscular sense is poor in left hand; cannot recognize a coin, knife or key. No paralysis in lower extremities. Movements good in all directions. Knee-jerks absent. No clonus. Pulse 60. No cardiac murmur.

Diagnosis.—Briefly the symptoms were as follows:

Severe pain over the occiput and back of neck. An uncertain gait; staggering, with a tendency to fall to the left. Hiccough, dysphagia, salivation. Paralysis of face and arm on left side, with ataxia and loss of muscular sense. Double optic neuritis and nystagmus. From the history of the case and the symptoms already enumerated, the diagnosis was made of "tumor of the cerebellum, involving the middle lobe."

March 21.—About 8 P. M., patient insisted upon rising up in bed and his wife assisted him. He no sooner assumed the upright position than he became very pale and died.

March 24.—Autopsy held fifty-six hours after death in the presence of Drs. Tygert and Phelps. Head only examined. Rigor mortis well marked. Nothing abnormal noticed about the external surface of head. No blood escaped on removing the calvarium. Bones of skull normal. Depressions of the pacchionian bodies quite deep. With the exception of a few slight adhesions along the longitudinal sinus the dura mater was normal. The subarachnoid fluid was increased in amount especially at base. No decided flattening of convolutions. Whole brain pale and soft. Vessels of pia mater quite prominent. Cortex and ganglia at the base of brain appear normal except for prominence of puncta vasculosa.

Cerebellum.—On the under surface of the left lobe of the cerebellum (Fig. IV.), occupying its inner third, extending over median line, pressing upon the medulla, fourth ventricle, vermis and spinal cord, is a tumor four inches long, three and a quarter inches wide and two and three-quarter inches in its vertical diameter. The tumor is irregular in outline, broken down in places and encroaches upon the ninth, tenth and twelfth nerves of left side.

Microscopically the tumor consists of a multitude of small round cells, in places showing myxomatous degeneration. Blood-vessels quite abundant here and there in the sections; deposits of blood pigment were present.

Remarks.—The sudden death in this case was probably

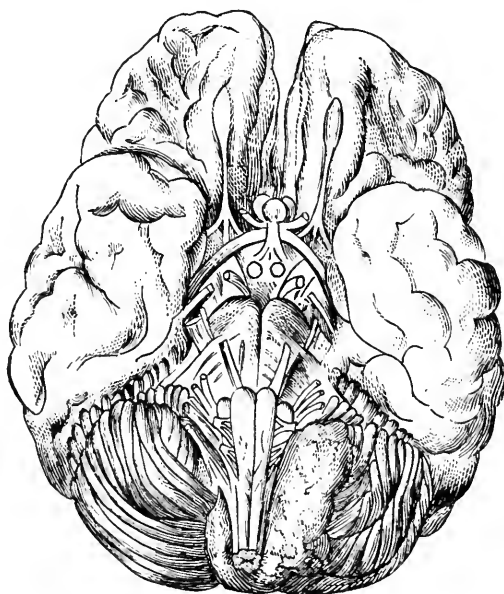


FIG. IV.

due to paralysis of heart and respiration caused by pressure on the pneumogastric.

From the situation and size of the growth it is interesting to note the absence of vomiting, any great excess of fluid in the ventricles, and convulsive seizures.

Society Reports.

AMERICAN NEUROLOGICAL ASSOCIATION.

Sixteenth Annual Meeting, held at Philadelphia, June 4th, 5th, and 6th, 1890.

(CONTINUED.)

A CONTRIBUTION FROM BRAIN SURGERY TO THE STUDY OF THE LOCALIZATION OF THE SENSORY CENTRES IN THE CEREBRAL CORTEX.

Dr. P. C. KNAPP reported a case in which there had occurred progressive dementia following a bullet-wound received twenty-eight years before. The patient was trephined and a portion of the cortex, most probably in the ascending parietal convolution, was removed. This was followed by inco-ordination, tactile anæsthesia, loss of sensibility to pressure, weight, localization, position, and motion. The sensibility to pain, heat, and cold was normal.

DISCUSSION.

Dr. C. K. MILLS thought the case was hardly convincing as a proof of the existence of the localization of sensory centres in the motor region of the cortex. The man had, previous to the operation, a condition of chronic degeneration of his brain. Then half an inch of variation in localization might have thrown the position of the lesion into what the speaker believed was a part of the sensory area of the cortex. These sensory centres probably surrounded the motor region and the median and lateral aspects of the brain, and this lesion might have involved the postero-lateral region. In a case of Dr. Lloyd's, half to one inch of the motor cortex was removed with some subjacent tissue. They had examined this patient many times with a view of ascertaining the existence or non-existence of tactile anæsthesia. There were present certain peculiar conditions, such as slight motor loss which simulated true tactile anæsthesia. The speaker believed in the distinction between the sensory and motor areas. He recognized, however, that we had few cases of reports of motor loss following lesions in the areas for sensation; the probable explanation being that there was almost instantaneous taking up of the function by the other hemisphere.

Dr. GRAY said it was extremely difficult to be certain as to the existence of a slight degree of tactile anæsthesia. The æsthesiometer was an unreliable instrument, and its

use was the more difficult when the patient was an uneducated person. It was difficult to make any fine diagnosis in the course of such a surgical operation. The question of the overlapping of the sensory and motor areas was at present one of mere speculation. At any rate, he did not believe that they were so precise in their outline that they could be located during surgical operations.

Dr. C. L. DANA said that Dr. Knapp had presented the positive and Dr. Mills the negative side of this question. It was possible to cut away the cortex and have no anæsthesia. He could not find any other than the motor area for tactile sensation.

Dr. KNAPP admitted the uncertainty as to the location of the lesion. He knew also that it was not easy to detect the minor forms of tactile anæsthesia; but in this case it had been marked, both before the operation and for some time after it.

ON CASES OF POSTERO-LATERAL SCLEROSIS, WITH SPECIAL REFERENCE TO THE PATHOLOGY OF THE DISEASE.

Dr. PUTNAM referred to a series of eight cases of similar character, presenting the symptoms of "combined sclerosis" of the spinal cord, which he had seen during the past few years, and reported four of them, in which he had examined the cord microscopically.

All the cases of the series, though differing in some respects, resembled each other as follows: All the patients were past middle life, and all were either anæmic or in a state of poor nutrition. The symptoms in all consisted in both motor and sensory disorders in all four limbs, sometimes associated with inco-ordination, sometimes not. The upper knee-jerk was exaggerated in all but two or three; in those it was absent. Tabetic pains were present in one case only. Anatomically, sclerosis was found in the posterior and lateral columns, varying in exact position. In almost every case the posterior change seemed the older and most intense. Besides the "typical" sclerosis, there was evidence of a more recent process, characterized by granule-cell formation and the breaking down of the nerve-tubes so as to form circular or oval spaces. This new process was developed on the borders of the older change. The gray matter of the cord was more or less affected, and the nerve-roots in about the same degree. The cases had all run a rapid course, terminating, after one to four years, in death, preceded by paraplegia due to non-inflammatory softening. Next to inherent structural weakness, as an etiological factor, came impaired nutrition and toxic influences. The importance was pointed out of recognizing and attempting to meet the

partial courses of the disease, of which several might be present at once.

As special stigmata of degeneracy in these four cases, the writer referred to the mental condition and family history of several of the patients, to the remarkably abnormal shape of the cord in one, the small size of the dorsal gray matter in another, and the presence of a second central canal in a third.

DISCUSSION.

Dr. S. G. WEBBER cited the case of a young girl who, from excessive walking, had developed decided symptoms of lateral sclerosis. There had been no lead; for arsenic no examination was made. Gradually this patient had recovered the use of her limbs.

Dr. PUTNAM, replying to Dr. Dercum, said there had been no serious involvement of the internal organs in any of these cases and no typical Bright symptoms. In one case there had been casts in the urine. The vessels of the spinal cord were all thickened, and there was weak heart, but no valvular lesion. These patients were so largely women, that he could not but assume the existence of some lesion from impaired nutrition and constitutional taint.

ON INGRAVESCENT APOPLEXY.

Dr. C. L. DANA read a paper with this title. He said that there were three sets of intra-cranial blood-vessels—those in the dura, those in the pia mater, and those in the substance of the brain. We had, correspondingly, three types of intra-cranial hemorrhage. The central hemorrhages were far the most common, and presented a tolerably uniform clinical type. There was one form, however, which seemed to have escaped critical attention, though it could not be excessively rare. In 1876 Dr. Broadbent had reported six cases of what he termed “ingravescent apoplexy.” In 1889 M. P. Puesch, of Montpellier, had also reported a case of the same character.

The writer had met two cases presenting the general clinical characters of ingravescent apoplexy, but was able to make an autopsy upon only one, of which the history was as follows: A woman was brought to the hospital on May 1st without any history. She was in a stupid condition, but not unconscious, and she was at first thought to be intoxicated. Examination showed, however, some hemianalgesia of the left side and slight hemiplegia of the same side. The right pupil was slightly contracted, temperature normal,

pulse tense. Next day the patient's mind was clearer; she answered questions, and recognized those about her. But the hemiplegia was very much worse, and the analgesia no better. Toward night she became more stupid, and finally comatose; œdema of the lungs developed. No contractures of the paralyzed side were noted. The temperature rose, and the patient died next day, May 3d.

At the autopsy the brain was found congested. Pressure over the supramarginal gyrus showed that there was a softened place beneath it. The brain was placed in boroglycerine and alcohol, and opened later by vertical sections. These showed a clot in the lateral ventricle, and some blood in the third ventricle. Beneath the supramarginal gyrus was a large hemorrhagic focus about an inch and a half in diameter. This extended forward and downward, cleaving the external capsule. The hemorrhage had finally extended downward and inward and broken into the lateral ventricle.

Puesch had attempted, on the slender basis of seven cases, to erect "ingravescent" or (as he called it) "progressive" apoplexy into a distinct type. This seemed to the author to be somewhat premature. The history of his case was not exactly like those of Broadbent's in respect to retention of consciousness, and the hemiplegia was relatively less marked. Yet, anatomically, it was one of the "cleaving" hemorrhages due to rupture of a posterior branch of a lenticular artery, and running the same course as was described by Broadbent. The hemianæsthesia seemed to the author to be a very distinctive point. Practically, the question came up as to whether, in such cases, trephining would be justifiable. In general, the idea of trephining for non-traumatic hemorrhage was not to be entertained at all. But in ingravescent apoplexy it deserved consideration, because here the hemorrhage was accessible, and because, unless some relief was given, it would surely break into the lateral ventricle and kill the patient. In all the reported cases, also, the patients were not old, were not syphilitic, and presumably had not extensively diseased arteries.

In reaching hemorrhages in these cases, the best place to trephine would be a little below and in front of the parietal eminence. The surgeon should then explore downward and forward, care being taken not to injure the terminal branches of the Sylvian artery, which were in this neighborhood. In cases of "ingravescent" apoplexy, surgical interference, if undertaken, should be before the blood broke

into the ventricles. This could be told by the sudden increase in the severity of the symptoms, and, if the blood was poured in rapidly, by contractures on the paralyzed side. The temperature changes were believed to be the same in the ingravescient as in ordinary apoplexy. The author trusted that the report of his case might excite the interest of others, and call attention to this apparently distinctive and fatal form of cerebral hemorrhage.

DISCUSSION.

Dr. MILLS had seen a number of cases. The clinical features were peculiar. Patients would be brought in in a condition of apparently incomplete unconsciousness, having the appearance of being conscious which was not real. The result of his experiments as to the best method for operative interference in these cases led him to think that the trephining, if done at all, should be several inches back from the temporal lobe along the junction of the second and third temporal convolutions. He believed that in some cases the blood when reached would not be found fluid. He thought the hemorrhagic focus might be reached by making an opening, enlarging it with some flat instrument and gradually removing the clot. One case of trephining for ventricular hemorrhage had been done. It seemed absurd, yet certainly cases of intra-ventricular hemorrhage had recovered independent of the question of trephining.

Dr. W. SINKLER said Dr. Dana had referred to the question of temperature. The speaker had recently had a patient die from enormous hemorrhage into the ventricle, and the temperature before death had been 108° , and half an hour before death $109\frac{2}{3}^{\circ}$.

Dr. WEBBER questioned the utility of trephining in these cases. In most the history was rupture. The force of the blood tore up the brain-substance, a considerable area of which was destroyed and infiltrated with blood. The hemorrhage continued, and then there was rupture into the ventricles. Opening the brain in such a case was apt to cause a continuance of the bleeding.

Dr. MILLS said the advantage of trephining was that it gave the patient a lease of one or two weeks, as the breaking into the ventricles did not take place until late. The bleeding stopped, and began again. By trephining and binding the bleeding point there was a chance of controlling it.

Dr. DANA said that trephining was purely empirical. He could not say whether it was good or not. He could not see any *a priori* reason against it because we changed

the condition of circulation in the brain, and surgeons might devise measures by which the hemorrhage might be stopped.

Dr. WEIR had made some experiments as to the site for trephining, described by Dr. Mills, and had come to the conclusion that it would be better in front of the fissure of Rolando.

HYPERALIMENTATION IN ACUTE DELIRIUM.

Dr. G. Guicciardi (*Rivista sperimentale di Freniatria*, xv., 465). Guicciardi has obtained in the treatment of acute delirium less unfavorable results than one usually observes in this disease; he only lost, out of eleven cases, four by death, while five completely recovered, and two became weak-minded. The treatment adopted by him consisted in isolation, removal of all sources of irritation, protracted luke-warm baths, with the simultaneous application of ice-bladders to the head and a few leeches to the mastoid process, as well as in revulsion to the intestine and ergotine injections. But he places a still greater emphasis upon the richest possible feeding, and indeed by methodic feeding, by means of the sound, every three to four hours with 350 c. cm. of the most nourishing fluid food.

MASSAGE IN MENTAL DISEASES.

Dr. Giuseppe Guicciardi (*Archivio italiano per le malattie nervose*, etc., xxvi., p. 393). Guicciardi has for some time employed massage in the treatment of mental diseases, and in general he is satisfied with the results. He has especially been successful in states of general depression, in stupor, and in exhaustive psychoses; the general condition improved in all cases and usually also the psychic relation. He recommends "general massage" of the body, and reserved a more special local massage for those cases, as hysteria, neurasthenia, etc., which are accompanied by disturbances of sensibility, neuralgias, contractures, displacements of the uterus, obstipation, etc. The massage should be continued long after the disease has disappeared; other methods, as hydrotherapy and electrotherapy, should by no means be neglected.

THE
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Original Articles.

DIFFUSE CORTICAL SCLEROSIS OF THE
BRAIN IN CHILDREN.¹

BY WILLIAM N. BULLARD, M.D., of BOSTON.

I DESIRE under this title to place certain cases of diffuse sclerosis in which the cortical layers of the cerebrum are the parts primarily or principally affected, and where the microscope shows the presence in these layers of a homogeneous ground substance, or of a finely fibrous network with a few spider-cells in the meshes, occupying the position of the neuroglia. In children but few cases in which this form of disease exists have been accurately described, and in our text-books the whole subject of diffuse sclerosis in children is still obscure.

That the form of cortical sclerosis or degeneration which I am about to describe, can be, in many cases at least, clinically differentiated from other forms of cerebral sclerosis, I feel confident. From ordinary lobar sclerosis and from the different forms of tuberous and multiple sclerosis, this affection can usually be distinguished. It is, on the other hand, closely allied to the conditions found in adults in general paralysis (paralytic dementia) and in chronic alcoholism.

The cases of this affection of which careful descriptions exist are very few.

¹ Read in abstract before the American Neurological Association in Philadelphia, June 5th, 1890.

In the St. Bartholomew Hospital Reports for 1879. Moore gives the case of a girl, five years' old. Previously in perfect health. Six weeks before her entrance into the hospital she fell twice. This was followed by twitching of the left side of the face and hemiparesis of the extremities on the left side. Ten days later occurred sudden right hemiplegia and aphasia. On admission to the hospital the pupils were widely dilated, insensible to light, no strabismus. She could not see, but could hear a little. She resented being touched, frowned and screamed; ground her teeth frequently. Did not control sphincters. Had slight clonic movements of all the extremities, stronger on the left than on the right. No loss of sensation. Shortly after entrance had two epileptic attacks, and later opisthotonos. Died about six weeks after entrance. The autopsy showed sclerosis of the cortex to a large extent; the white matter was unaffected. Microscopically, increase in neuroglia, diminution in number and possibly in size of nerve cells.

Schmaus case is somewhat analagous, but we are inclined to think should rather be classed among the lobar sclerosis, in which the process may be very diffuse. It occurred in a girl three years old. The pathological condition was "Diffuse sclerosis with enormous atrophy of the cerebrum, especially of the convolutions. Hydrocephalus internus et externus ex vacuo. Pachymeningitis interna. Hydrocephalus externus. Atrophy and sclerosis of the anterior columns of the cord, microscopically. All cortical layers diminished, but easily distinguished. Principal change is in the existence of a very thick net-work of fine fibres, lying between the ganglion cells. The ground substance in the white matter is likewise a fine net-work."

My own case, which was quite remarkable in some respects, is as follows:

The patient, when first seen, April, 1887, was a boy 13 years old, a native of Boston. The family history, which was carefully enquired into, showed nothing remarkable. No neurotic tendency.

The patient was born at full term, the labor was natural, and no instruments were used. His general health has

always been excellent. He learned to walk when 19 months old. Has always been bright and intelligent, wrote a good hand, and at the time of his accident was in the 5th class of the Grammar school.

In August, 1886, he had a fall (was said to have been knocked down) and struck his head against the curbstone, making a wound over the R. parietal bone, the scar of which still remains. He did not, however, complain of any pain in it, though it was somewhat tender, and his whole head has been sensitive all winter. At the time of the accident, he was perfectly healthy, and he was promoted in school after the injury, but his mother believes that after it, he was stupid at school, and did no work.

The first thing peculiar noticed about the patient was three weeks after the accident, when he forgot the baby whom he had taken out of doors. He was accustomed to take the child out, and was very fond of him. Two months later, he lost himself one day, and could not be found until the day following, having spent the whole night out. He could not tell people where his home was, nor find it himself.

Since the accident, he has gradually lost his memory, become forgetful and lost interest in external objects.

In the beginning of his illness it was noticed that he kept his tongue in constant motion in the R. side of his mouth, but this ceased some time ago. He has become awkward on his feet and cannot use his hands, as he used to do.

Physical strength and general health, excellent. Has had apparently slight difficulty in sight.

Present condition (April, 1887): Physically well developed and in good condition. Is very forgetful and childish, weak-minded. Will come across the room to see a watch, and smiles at it like a baby. Sits quiet much of the time with a vacant expression. Understands fairly well what is said to him. At home, likes to sit by the window and watch what goes by. Can read the name on a wagon, but does not connect any idea with the words, and cannot even do this now as well as he could at first. Remembers the boys who

were in his room at school since his injury, but nothing of those who were there previous to his injury. Speech slow, indistinct. Occasionally omits words. Never uses the wrong word. Can use neither hands nor feet well, now. L. hand the more helpless.

Physical Examination: Of average size. Muscular development good. Head of normal size and shape. Scar 1 1-2 inches long over R. parietal bone. R. abducens seems weak. Tongue protruded straight, but seems to move more readily to L. Cannot be induced to squeeze with hands, but can pull fairly well. L. hemi-paresis. Clonic spasms L. arm and face. Incoordination of L. upper extremity and probably of L. lower extremity. Stands with legs apart, as in hypertrophic paralysis. Waddles in walking, which is difficult. Sensation is possibly diminished, as he is said not to complain of burns or wounds on hand. He declares, however, that he feels the pricking of a pin everywhere on the legs readily, and says "prick" when he is touched with it. Skin reflexes on trunk and plantar reflexes excellent. Knee-jerks alike, exaggerated.

On the 24th of April, entered the Carney Hospital. His eyes were examined by Dr. Standish, who reported R. eye veins tortuous, exudation along the arteries and atrophy of choroid. L. eye vessels all small. Neuro-retinitis of both eyes, most severe on the right.

Child seems happy. Has a habit of repeating the emphasized words of a sentence spoken to him, and also repeats his own words. Temperature morning and evening, normal.

At this time he was seen in consultation with Dr. Burrell, and it was decided to trephine at the seat of injury in the hope of finding some source of irritation, it being evident from the child's condition that he was constantly growing worse, and the prognosis being distinctly unfavorable, unless some change could be induced shortly.

On the 27th of April, therefore, the patient was etherized. Slight clonic spasm of L. upper extremity, as patient enters under the influence of ether. An oval, somewhat excavated cicatrix, 1 1-2 inches long by 3-8 inch. in width.

is found externally, situated s 3-4 inches to the right of the sagittal suture, and somewhat posterior to the line passing from one auditory meatus to the other over the vertex.

The cicatrix was probably over or just posterior to the median portion of the ascending parietal convolution.

The skull was trephined by Dr. Burrell over the cicatrix and a large disc removed. The dura-mater appeared normal, and there was no bulging. A trocar inserted forwards and somewhat inwards, met with no resistance. The bone was replaced. The external wound was closed with cat-gut sutures. The wound healed readily, and the patient was discharged from the hospital.

There was a slight, temporary cessation of the clonic spasms, but no permanent improvement. The only change noted while in the hospital was, that he lost the power of speech almost entirely. Screams considerably.

On the 22d of June, two months after the operation, I saw the patient at his home. He was found lying on a mattress placed on the floor. He is unable to get up by himself, but when standing can walk from one room to another. After first leaving the hospital, he screamed almost constantly for two days ; now screams at intervals, if at all excited, and sometimes without apparent cause. Cannot talk at all now. No change in the special senses noticed. There has been, since April, an increasing incoordination and paresis, involving all the extremities, but more markedly those on the L.

August 8, 1887. On the whole, no worse than at last report. Does not scream much, except when changed after wetting clothes. There has now been incontinence of urine for some time.

September 28. Cannot speak. Cannot sit upright. Cannot use hands or feet much. Incontinence of urine and fæces. Laughs and makes inarticulate sounds when spoken to. Recognizes mother. Grinds his teeth constantly. Inclines to pull things to pieces. Sleeps quietly, very lightly.

November 7. L. side now (completely) paralyzed.

Shortly after this, being utterly helpless, and his mother no longer being strong enough to lift him, was committed by Dr. Jelly and myself, and sent to the Boston Lunatic Hospital. There he died a fortnight or so after admission in a condition of extreme dementia.

The autopsy was performed by Dr. Gannett, and it is published as No. 77 in the Forty-ninth Annual Report of the Superintendent of the Boston Lunatic Hospital for the year 1887.

"Male, 14 years. Autopsy twenty-nine hours after death. Body well developed and fairly well nourished; slight lividity of dependent portions; rigor mortis present. There was a horse-shoe shaped scar over the right posterior parietal region; a circular disc of bone had been removed and replaced, and it had become firmly adherent to the surrounding bone; dura not remarkable. The brain did not quite fill the cavity of the skull, and weighed 1,260 grms.; sulci wide; pia showed a slight degree of thickening and opacity; the meshes contained a moderate amount of thin, clear fluid; vessels at the base and fissure of Sylvius showed nothing remarkable. Both lateral ventricles were very greatly increased in size. The surface of the ventricles was quite smooth and quite tough to the sense of touch. Ependyma of fourth ventricle markedly granular. Brain substance in general, pale and very tough. Both nuclei caudati were unusually thin; the other basal ganglia not remarkable; pia readily separable from brain substance. Organs of thorax and abdomen showed no variation from the normal."

DIAGNOSIS.

Wound of earlier trephining.

Œdema of pia.

Chronic lepto-meningitis.

Atrophy of Brain.

Chronic internal hydrocephalus, secondary to atrophy of brain.

Chronic ependymitis of fourth ventricle.

No evidence of compression or obstruction of vena Galeni.

Microscopical examination. Pia showed a slight degree of thickening from growth of fibrous tissue.

BRAIN.—First layer of cortex showed the finely fibrous net-work with a few spider cells in the mesh, due to atrophy of the nerve fibres and an increase in the neuroglia. There was a slight degree of nerve cell infiltration of the adventitial sheaths of the blood vessels in the cortex. Beyond this, nothing abnormal was observed.

The interest in this case consists in part in its rarity. Very few cases of primary diffuse cerebral sclerosis in children of this age, have been reported. There are numerous examples of cerebral sclerosis, either congenital or occurring in the earlier years of childhood, but they apparently differ somewhat in character from mine. Pathologically, too, I believe them to be distinct. Secondary cerebral sclerosis due to hemorrhage, tumor, chronic hydrocephalus or other cause (embolism or thrombosis, loss of extremities) is common.

Taking simply Moore's case and my own as the type of this form, the clinical picture presented is as follows: The onset of this disease was in both cases ushered in by a fall from which the commencement of the symptoms is dated by the relatives. In a varying period after this, there appears some intellectual impairment, shown at first simply as obtuseness, general dulness and loss of memory. At about the same time temporary clonic spasms begin to occur, perhaps first affecting the face or tongue, but gradually involving the extremities, those on one side of the body being less affected than those on the other. These convulsive attacks are for a time unaccompanied by loss of consciousness, though later in the disease true epileptic seizures may occur. Nearly simultaneously with the development of the active motor symptoms, there occurs a loss of motor power, sometimes appearing suddenly as a hemiplegia; sometimes very gradually, but usually involving the extremities on one side especially, later those on the other side being likewise affected. In addition to this loss of power in the extremities, we sometimes find a very marked diminution in the power

of co-ordination or control, likewise somewhat hemiplegic in distribution, which in the earlier stages may even mask or conceal the paralysis. The speech is soon affected, either becoming slow, thick and difficult, or aphasia may occur in connection with a right hemiplegia. The loss of power in the extremities increases as a rule progressively and gradually involves the sphincters, causing incontinence of urine and fæces. It has, however, never become absolute in any case yet reported, but has reached such a point that the patient was incapable of sitting up alone or of making any more than a slight motion with any extremity. As the disease progresses the spasmodic manifestations incline to increase in frequency and severity, and we may have epileptoid seizures and opisthotonos.

In spite, however, of the progressive character of both the motor and intellectual symptoms, we find sensory symptoms almost absent. There are no anæsthesiæ or paræsthesiæ apparent. No pain is complained of and no definite loss of sensation has in any case been determined. Unfortunately we know little or nothing in regard to the condition of the muscular sense. Neuro-retinitis or optic neuritis occurs early. The other special senses seem usually unaffected, but as the progressive failure of intellect becomes more and more marked, no absolute determination of their condition can be made. Like the motor, the mental symptoms progress gradually, but steadily.

In the patient under my care, the mental condition was throughout that of a simple, rapidly progressive deterioration of all the mental powers, a pure dementia, uncomplicated by any intervals of excitement or violence. There was no evidence of melancholia; on the contrary, the patient seemed in excellent spirits, happy and contented, until he reached the more advanced stages of the disease. No hallucinations or delusions were at any time found. The mechanical grinding of the teeth, which became nearly constant at times, and the at least partially unconscious screaming, which in the later stages was sometimes continued for hours, were rather evidences of unconscious or semi-con-

scious reflex actions than of any conscious mental or intellectual condition.

The symptoms of this affection, in short, are those of a gradually progressive mental deterioration, uncomplicated by hallucinatory or delusional conditions and a gradually progressive loss of power, involving all the extremities, the trunk muscles and the sphincters, a loss of speech and loss of sight, accompanied by the symptoms of motor explosion, clonic spasms, tonic spasms, and eventually epileptic seizures ; while, on the other hand, we find an almost total absence of all sensory disorders. In my case, we find a very marked co-ordination of the extremities. In neither of these cases was there either tremor or nystagmus, both of which were present in the case of Schmaus.

A case related by Turnbull and by him placed under the title of general paralysis, commencing in a boy of eight, presents symptoms very similar to those of diffuse cortical sclerosis. In this case there was less apparent prominence of the motor symptoms, although the patient had an attack of hemiplegia, which seemed to act as the starting-point on which the later dementia was engrafted. The duration of the affection in this case was 10 years. At the autopsy was found chronic pachymeningitis, chronic lepto-meningitis, external hydrocephalus, ependymitis and atrophy and flattening of the cerebral convolutions.

McDowell's case in a lad of 18 is clinically very similar. There is no mention of any meningitis at the autopsy, but there was atrophy and induration of the whole of both cerebral hemispheres, and patches of softening in three places.

We now come to the question of diagnosis. Can this affection be accurately diagnosed during life and distinguished from other cerebral affections, with somewhat similar symptoms ?

The most important practical point is to determine in any case whether we have to deal with a diffuse process, or whether the symptoms presented may possibly be caused by some process more or less localized. In certain cases this is by no means an easy matter to determine. Where

the explosive symptoms predominate and the paresis is slight or unilateral, and especially where, as in my case, a cicatrix exists over the motor region on the side opposite to that of the hemiparesis, the possibility of a localized starting-point cannot be excluded. It is the possibility of a localized lesion or new growth, accompanied by a chronic or subacute inflammation of the meningeal membranes, which sometimes renders an absolute diagnosis impossible. A cerebral tumor uncomplicated with meningitis would be unlikely to produce this combination of symptoms.

In regard to multiple sclerosis, the differentiation from congenital cases and from those forms occurring in epileptic idiots is simple, and may be determined by the history. A number of cases have been reported, however, by competent observers of multiple sclerosis in children, in which the symptoms more or less clearly resembled those of the same disease in the adult, but so far as I have been able to discover, that of Schüle is the only one in which the diagnosis was substantiated by the autopsy. At the present time, therefore, we can only say that we should expect in uncomplicated cases of multiple sclerosis in children a greater similarity to the symptoms of that disease as shown in grown persons than to those of diffuse cortical sclerosis. A slow development, beginning with nystagmus, tremor and paresis, with a distinct subordination, at least until the later stages of the disease, of the mental symptoms.

From the congenital or early forms of cerebral atrophy and from all forms of partial or lobar atrophy (excluding multiple sclerosis) the history of the case is usually sufficient to exclude. But, in any case the non-progressive character of the disease in these cases forms a distinction.

In cases of primary hydrocephalus, so-called, the symptoms are usually of a very different character from those just detailed. The presence of rachitis would of itself suggest this affection. As a rule the mental symptoms are developed late, if at all, and the symptoms of cerebral compression are prominent at some period of the disease. It is, perhaps, scarcely needful, however, to mention that hydrocephalus occurs as a secondary condition in diffuse sclerosis.

The conclusions that I would draw provisionally are, that there exists a form of diffuse cerebral sclerosis in children in which the cortical layers of the brain are more especially affected, and which is clinically distinguished from the other forms by its appearance in healthy children, either without known cause, or after traumata, by the steadily progressive character of its symptoms, and by the especial prominence of the gradually increasing dementia, which finally reaches an extreme degree without a correspondent loss of motor power, and while the sensation is comparatively unaffected. The pathological condition found is pathologically undistinguishable from that found in adults in general paralysis.

BRACHIO - FACIAL MONOSPASM, FOLLOWING
PROBABLE EMBOLISM, WITH CONSEQUENT
DEGENERATIVE CHANGES IN BRAIN. AND
LOCALIZED MENINGITIS—DEATH FROM APO-
PLEXY.¹

BY MORRIS J. LEWIS, M.D., OF PHILADELPHIA.

MRS. —, æt. forty-eight. Patient married for many years, but childless. No history of syphilis. At age of twenty-one years (in 1862) she had a severe attack of inflammatory rheumatism, seriously affecting the heart; no serious trouble followed this until nineteen years later (in 1881), when, after a severe attack of cardiac palpitation which lasted several days, during the latter part of which she had a right-sided headache, she became paralyzed on the left side; the paralysis was not complete, was preceded by some indefinite face movements, was not accompanied by any loss of consciousness and completely passed off in thirty-six hours, the face remaining paretic longer than any other part. Occasional uncomfortable sensations in her head followed this attack, and in June, 1882, after an attack of headache and vomiting, she was seized in her sleep with a convulsion, the character of which is unknown; no paralysis succeeded this. In August, after having had two convulsions a short time before, she had a series of terrible convulsions, seventeen in all, lasting twenty-four hours, consciousness not being regained in the interval.

Each of these attacks began with twitching of the left face, turning of the head and eyes strongly to left and twitching of the left hand and arm, after which the convulsions became general.

¹ Presented to the American Neurological Association at its annual meeting held in Philadelphia, June 4, 1890.

Complete paralysis of the left side of the body, lasting for several days, followed these seizures, and she was unable to walk for two weeks.

During the winter of 1883-84, she had numerous attacks resembling *petit-mal*, in which she would have a drooping of the left eyelid, and a tendency to lose consciousness. Subsequently the fully-formed attacks appeared again, and returned at intervals of about seven weeks.

Such was the history of the patient when she came under my charge in 1886. She was having convulsions at irregular intervals. Her heart was hypertrophied and irregular in action. Apex beat in fifth interspace one inch to the left of the nipple line. There was a soft systolic murmur loudest over sternum, but also heard loudly at second right cartilage. At apex there was a soft low systolic murmur of different pitch, which was conveyed faintly into axillary line. The lungs were healthy. The urine was light amber in color, acid, sp. gr. 1014. A trace of albumen existed, but only to picric acid test, and not to nitric acid. Microscopical examination showed a few pus cells and one granular cast.

Further examination of patient showed that there was no paralysis, and no wasting or rigidity. The knee-jerks were normal; sensation everywhere good, speech was in no way interfered with, but intellection seemed a trifle blunted. The eyes were examined by Dr. G. E. de Schweinitz, who reported: Normal central vision by the correction of a low degree of hypermetropia, three degrees of insufficiency of the external recti. Oval optic nerves, scleral ring marked all around and the central lymph sheaths full; slight retinal haze, no other changes. A second examination five months later (June, 1887) showed that the field was normal for form, with slight concentric contraction for red and blue.

In May, 1887, I was fortunate enough to see an attack from beginning to end. The movements began apparently simultaneously in the left hand and face, as follows:

Twitching of the left forefinger in flexion, then twitching of the other fingers and wrist; at the same time there was twitching of the left face with movements of the lower

jaws, the eyes and head were convulsively jerked to the left; during this part of the attack consciousness was retained, but was subsequently lost as the attack became general. The tongue was now bitten for the first time.

This seizure was of short duration and was followed by a transient paralysis of the left hand, arm and face.

By bromides freely used, and nitrite of amyl given by inhalation when the premonitory signs appeared, the seizures were kept in abeyance until May, 1888, when she had a severe attack.

During 1888 she had numerous seizures, which always began with severe stinging pain in the right temple, accompanied by flashes of light, resembling the flames of several large candles, although this last symptom occasionally occurred without a convulsion following.

Pricking in the left arm and leg were frequently complained of in the prodromal period which lasted almost exactly two hours. The convulsion when not prevented by treatment almost invariably occurring during the last five minutes of this period. Pin-head sized extravasations of blood under the skin of face and left arm frequently occurred in consequence of the severity of the paroxysms.

The signal symptom always appeared in the face and hand, although sometimes the one and sometimes the other seemed primarily affected.

Paresis, more or less pronounced, always followed the attacks, and was limited to the parts first convulsed.

Twice after the usual premonitory symptoms, paresis supervened *without the previous convulsive stage*.

In 1889 the seizures continued about the same. Menstruation, which previously had been fairly regular in its appearance, became irregular, and finally ceased; no positive relation could ever be discovered between the menstrual periods and the attacks. During this year the patient began to become melancholy and had occasional hysterical attacks, in which she would scream, throw herself about, and kick and strike her attendants. Once or twice she disappeared and was found shut up in a dark closet; she said she was tired of life, and talked of committing suicide.

Frequent examination of the urine gave a sp. gr. varying from 1014 to 1020 with a normal amount excreted

Albumen was always present in small amount, pus cells could always be seen in moderate quantities, and occasionally a granular or hyaline cast could be found. Oxalate of lime and uric acid crystals were frequently present.

In October, 1889, the condition of the eye-grounds, as reported by Dr. G. E. de Schweinitz, showed absolutely no changes over former reports, except slight failure in accommodation due to advancing years. No record of the fields of vision were made at this time.

In November, after a period of melancholy and depression, she had a severe general headache, and towards the end of the month this was repeated; it was then discovered that there was a marked difference in the pulses in the two radials, the right beating very feebly while the left was strong; there was no pain in the arm, and the color appeared normal; the right leg was awkward and felt heavy to the patient, and the right posterior tibial pulse was weaker than the left. The heart was irregular, beating from 72 to 90 times a minute, and the auscultatory signs differed markedly from the usual condition, as previously noted. A sharp, high-pitched, short first sound existed at apex immediately followed by a long low murmur, the radial pulse following the short sound and preceding the long murmur by an appreciable interval. A decided thrill accompanied the first sound at apex. In the axillary line the murmur was not so clearly heard as at apex. Great restlessness existed at this time, and the patient was given a small dose of hydrobromate of hyoscin ($\frac{1}{200}$ grain), and went to sleep to awake in twenty minutes completely paralyzed on the left side and with a violent stinging pain in the right temple. The nurse was in the room all the time, and is certain there was no convulsion and that she was not paralyzed when she went to sleep. The patient awoke perfectly conscious and could talk, but the speech was thick. When seen by the writer half an hour later there was complete paralysis of the left arm and leg and almost complete palsy of the left face; the tongue was thrust out to the left, but there was no paralysis of the ocular muscles.

In a few minutes the paralysis of the left face markedly diminished, and she could close left eye and depress the angle of mouth, and the tongue was protruded in a straight line; power to slightly move the left leg returned for a few minutes, but again disappeared. Strong voluntary movements of the right side caused associated movements in the paralyzed arm. Left lateral homonymous hemianopsia was now found to exist, the blindness passing the fixation point—the left pupil was larger than the right, and “Wernicke’s pupillary reaction” was present in the left eye, but not in right. These symptoms were corroborated a short time later by Dr. G. E. de Schweinitz.

Sensation was greatly interfered with, a prick on left face or hand being localized as a pain in the left leg, while a prick on the latter was fairly well localized.

The temperature in the right axilla was found to be 97° , while that of the left was 98° .

Later in the night the circulation became very feeble, the pulse being scarcely perceptible in either carotids, or in the femorals, or in the right wrist, while the pulse in the left radial remained fair.

The next day, November 24th, *the only pulse that could be detected was that of the left radial.*

Dr. H. C. Wood saw her in consultation with me and diagnosed “Multiple thrombi closing the various vessels, and plugging all the branches of the right middle cerebral artery.”

Careful examination failed to detect any atheroma of the superficial vessels. Temp. right axilla, $98\frac{3}{4}^{\circ}$; of the left, 100° . Respiration labored, 40–48 per minute. The next day, November 25th, *pulsation could be plainly felt in vessels of right side of body*, the right femoral pulse being very much retarded. Left side of body hotter than right. Abdomen being particularly hot.

On the 27th patient was quite bright, talked rationally, and recognized odors. The hemianopsia remained the same. She could read perfectly, write at dictation, and express her wishes in writing, but was found to be deaf in the left ear; this was never noticed before.

Sensation was much better in hand, but complete paralysis of the leg and arm still persisted. Pulses could be felt in all the vessels, but right side were more feeble than left.

Knee-jerk feeble, no clonus; slight rigidity of *right* tendoachillis. Urine, sp. gr. 1024, very acid, trace of albumen, no sugar; pus cells in moderate quantities, no casts. On November 25th, Cheyne-Stokes type of respiration was noticed.

On November 29th the pulses in the two posterior tibial arteries were noticed to be *irregularly asynchronous*—that is, they not only did not beat together, but the two pulsations apparently bore no relation whatever to each other; this was noticed by her husband, who likened the pulsations to the beating of two clocks with pendulums of different lengths.

The peculiarity of pulses noticed on the 29th did not obtain at the next examination on November 30th. In December the patient became very much excited and continually cried out in a monotonous manner, all idea of modesty appeared to leave her, and she continually threw all the bed-clothes off, the right arm and leg being in constant motion.

On the 25th she had a slight convulsion, and she died on the 26th.

Autopsy.—Forty-four hours after death: head only allowed to be examined; time of examination limited to one and one-quarter hours, and under peculiarly trying circumstances; nothing but diseased area in cortex was allowed to be removed.

Skull quite thick; dura-mater adherent firmly for a small space at bregma. But little fluid escaped on removing brain. Base of skull normal. Brain: veins engorged, no basilar meningitis; some slight pachymeningitis at convexity. Circle of Willis presented no spots of atheroma. The right middle cerebral artery plugged with a dark clot from bifurcation back almost to carotid, passing the branches entering the anterior perforated space. A large area of softening of the gray matter existed as follows:

Upper convolution of right tempered lobe almost diffuent, supra-marginal gyre very soft; also softening anterior

to this as far as the fissure of Rolando, most marked softening towards supra-marginal gyre. At base of ascending frontal convolution the pia-mater was adherent over a space as large as a quarter of a dollar, and just anterior to this there was a spot where the convolutions were atrophied and sunken below the level of the brain; this portion was removed for examination (See Fig. I). A large

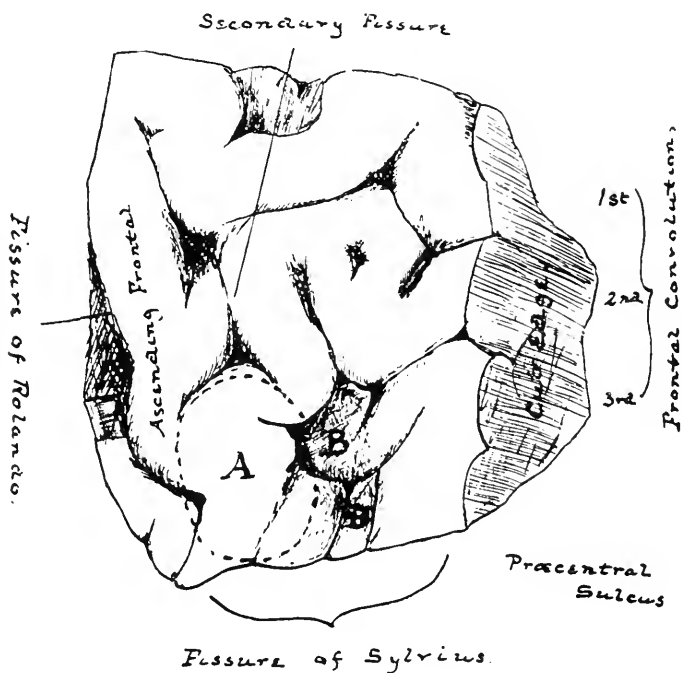


FIG. I.

hemorrhage existed just beneath the gray matter of cortex, extending from beneath the supra-marginal gyre forward to fissure of Rolando; the posterior portion far advanced into formation of pus, almost diffuent, darkish brown in color, extending deeply and cutting the radiating fibres from cuneus, which was perfectly normal in appearance; the anterior portion of hemorrhage was evidently of more

recent date than the posterior portion, as it was dark red in color and not so far degenerated into pus. No ventricular effusion. Large ganglia at base ruptured on right side. Nothing else abnormal was noticed.

Dr. Allen J. Smith, who kindly made the microscopic examination for me, reports as follows :

" The small section, a transverse section of the atrophied convolution, exhibits under the microscope evidences of chronic inflammation, particularly to be seen in the thickening of the vessel coats, and the obliteration (more or less) of the perivascular spaces by formed and embryonal connective tissue. The substance of the convolution is in varying degree infiltrated with formative cells in various stages of transformation into fixed connective tissue. The cellular nervous elements do not, however, so far as noted, escape staining well, and are not granular or especially atrophic or changed."

" The larger section from the position of the adherent membrane shows the same evidences under the microscope of chronic inflammation. The membrane is thickened, to some extent, due to nerve layers apparently on the inner surface. The adhesions are in consequence of the extension from or into the brain substance of the inflammatory change, the convolutions being infiltrated with formative and fixed cells, and the vessels injected and their walls thickened—the perivascular spaces being here also obliterated. There are numerous blood-cells infiltrated throughout the brain substance. Beneath the membrane are several foci of hemorrhage of slight size."

Iodide of potassium was tried twice, but iodism was produced long before any benefit could be expected, and while the dose was still small. Arsenic was administered to keep in check the tendency to the bromide eruption.

Nitrite of amyl at first seemed to have a good effect in breaking up an attack, but later in the case failed to do good. In the early part of 1887 I advised trephining and removal of the discharging lesion, which I localized at the point of union of the lower portion of the second frontal

convolution and the ascending frontal (see Fig. II., drawn at time), as this point would account for a discharging lesion affecting the hand, face and centre for turning of the head to the left. I should, as the autopsy proved, have placed the lesion a little lower down on the ascending frontal, as the face, particularly in the earlier convulsions, was more seriously implicated than the hand, although this condition was not so patent later. The localization was sufficiently accurate for operative interference, which was, however, refused. My opinion was that the starting point of the trouble was probably a small embolus causing the primary paralysis and setting up secondary degenerative

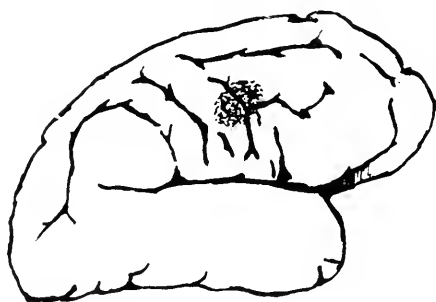


FIG. II.

changes with meningitis, which acted as a focus of irritation, resulting in a discharging lesion causing the brachio-facial monospasm. The possibility of the lesion being syphilitic was naturally borne in mind. Whether an operation for the removal of the diseased brain tissue would have resulted in a cure of the convulsions, and thereby have prevented the plugging of the middle cerebral, and the accompanying hemorrhage which caused her death, is a matter of conjecture. The serious organic disease of the heart would certainly have made anæsthesation a dangerous procedure; but I have always regretted that the operation was not performed.

My diagnosis of the final attack as opposed to that of Dr. Wood's was that there was a large hemorrhage into the internal capsule, cutting both the motor and sensory

fibres and probably catching the radiating fibres from the cuneus, as this would explain the paralysis of motion and of sensation and the hemianopsia, which plugging of the middle cerebral artery, however extensive could not do, although the autopsy proved this condition to co-exist with the hemorrhage, which by the consequent softening of the upper convolution of the temporal lobe probably explains the deafness in the left ear.

Whether the plugging of the middle cerebral artery which occurred shortly before death was primarily due to a small embolus, with consequent thrombosis, or whether the latter was primary, cannot be told easily at this late date, as so much organization of the plug has taken place, but it seems likely, on account of the previous circulatory disturbances, to have been a thrombus which produced sufficient damming back of the blood current to cause the rupture of the vessel with the consequent hemorrhage.

I was at the time, and am still, at a loss to explain the peculiarity in the pulses, which varied from day to day and even from hour to hour, no theory of thrombosis to my mind being at all satisfactory; unfortunately, no examination in this direction after death was allowed.

May there not possibly be some centre in the brain which controls the innervation of the arterial system, and which being irritated might give rise to such symptoms. The irregular a-synchronism of the two sides being the symptoms to which I especially allude.

Other points of interest are as follows :

Flashes of light as a prodromal symptom of a brachio-facial monospasm, the attacks of paresis without the preceding convulsion and Wernicke's pupillary reaction on one side only.

It was impossible under existing circumstances to obtain synchronous tracings of the two pulses. The tracings which were obtained were unsatisfactory, and showed nothing but the irregularity of rhythm. The temperature of the left axilla, was generally higher than the right, although on several occasions it was the same, and even much lower.

A CASE OF TUBERCULAR MENINGITIS, WITH AUTOPSY.¹

By WILLIAM BROADDUS PRITCHARD, M.D.,

Lecturer on Mental and Nervous Diseases, New York Polyclinic.

I DESIRE to present for the consideration of the Society the following history, with the brain, of a patient who died of tubercular meningitis on the 20th of September last. The symptomatic observations made during the progress of the case are almost absolutely accurate, even to the minutest details, a fact which should add materially to the value and interest of the history. Records were made for ten days previous to death, of temperature, pulse, and respiration, at three-hour intervals and the nurse, having been thoroughly posted in advance, was constantly on the *qui vive* for any symptoms which were to have been expected. A summary of the salient points in the history is as follows:

The patient, a Scotchman by birth, thirty-eight years old, was a man of delicate physique, small in stature, and weighing about 110 pounds. In temperament he was typically Scotch, reticent, nervously sensitive, and with great tenacity of purpose. Thoroughly educated in his youth, he had continued all his life a student. By occupation he was an expert designer and manager for a large lithographing establishment in this city. During the past fifteen years he had suffered from four or more pulmonary hemorrhages, the last about fifteen months ago. He had never had syphilis. At the time of the occurrence of the last hemorrhage he consulted Dr. Loomis, Sr., and was advised to go to Colorado at once, his case being pronounced one of phthisis. To leave New York at that time meant the overthrow of cherished business plans and great

¹ Presented, with specimen, to New York Neurological Society, Oct. 7th, 1890.

financial loss. The other alternative was not more reassuring. The dilemma proved a source of great mental worry and anxiety, which, with extraordinary, though largely assumed responsibility in business and close application to work, brought on a condition of apparent cerebral neurasthenia. Thirteen months ago he began to be troubled with headaches and insomnia, both of which gradually increased in degree. During the past five months his wife had been absent in Europe, awaiting the results of her first confinement, though married eight years. Anxiety as to the outcome, his wife being far distant in case of danger, was a factor largely increasing his mental worry, and a stranger in a strange land, having no confidants, he was forced to a dangerous self-communion.

At the time of my first visit, September 2d, I found the patient sitting in a darkened room, with the attitude and expression of utter mental exhaustion. The only symptoms complained of, in addition to the two mentioned above, were a slight nausea (no vomiting), a feeling of intense prostration and constipation. He stated that three hours in the twenty-four represented the maximum of sleep obtained for several weeks previously. The headache or pains he located in the region of the occiput, the upper neck and the brow. There was no corresponding pain on pressure or percussion in either region. This one symptom (headache) almost entirely monopolized the patient's attention. Physical examination showed an area of disease in the right lung at apex, an enlarged liver, a coated tongue, and nothing more. He had not had any cough, night sweats, or fever, and his weight had remained stationary for a year. The pulmonary trouble had been, therefore, for some time inactive. The temperature, pulse, and respiration were normal. A tentative diagnosis of cerebral neurasthenia was made, I being unable, at that time, to decide that the symptoms were due to organic disease. Two days later I saw the patient again, this time at my office, he having so far improved under symptomatic treatment as to have returned to his duties, I made an application of galvanism to the brain at this time (1.5 miliampères

for three minutes) with decided, though temporary, relief of the headache. Although the patient was directed to call again on Sunday (September 7th), he did not do so. When reminded of his engagement with the doctor by one of his household, he remarked: "What doctor?" "Dr. Pritchard," was the reply. "Who is Dr. P.?" he asked. His temporary confusion of mind clearing up in a few moments however, he requested that I be sent for and greeted me with his usual courtesy, and in an hour's conversation subsequently, upon various subjects, he showed a degree of brilliancy in his conception of ideas and of logical accuracy in argument which was beyond cavil. Upon being questioned he stated that he had noticed transient conditions of mental confusion, with inability to systematize his ideas and a limitation of his vocabulary. His manner, unless specially aroused by some unusual mental stimulus, was decidedly apathetic and melancholy. He sat in a darkened room by preference, although there was no decided photophobia. His temperature by mouth was still normal; pulse 72. The patient's condition remained practically unchanged up to Wednesday, September 10th, when, for the first time, the temperature rose above normal, the thermometer registering 101.4° F., pulse 76. Vomiting also occurred for the first time, the emesis being slight and consisting apparently of a simple regurgitation. It did not recur. The patient's mental state was one of increased confusion and apathy, possibly due in part to the action of thirty grains each of sodii bromid. and sulfonal, with seven and a half grains phenacetine, taken in two doses the previous evening, the result being ten hours sleep and a diminution of the headache. The diagnosis of tubercular meningitis, already suspected, was no longer considered a matter of doubt, and a prognosis given accordingly.

I shall confine my report of the subsequent clinical aspects of the case to a brief resume of the more essential particulars. The highest temperature observed, from the date of onset of febrile symptoms up to within twelve hours of patient's death, was 102.8 on the third and fourth days; the lowest 99.8 on the sixth day. The average temperature,

up to within twelve hours of death, taken at three-hour intervals, was 101.4. Respiration varied from 26 to 36, and was almost entirely abdominal for twenty-four hours before death. The pulse was typical, ranging from 60 to 70 until towards the close, when it ran quickly to 130, 160, and beyond. The first symptom indicating a paretic state was constipation, which persisted for a week in spite of varied and persistent efforts to overcome it, elaterium, with massage of the abdomen for an hour, finally producing the desired effect. A mild degree of right ptosis appeared on the fourth day and remained until within forty-eight hours of death, when it disappeared. It was at no time marked. A loss of the light reflex, with immobility of the pupil, was noticed about the same time; but no strabismus appeared during the entire progress of the case.¹ Some stiffness of the neck and a tendency to burrow in the pillows appeared early, but there was never a condition approaching opisthotonos. On Saturday, the fourth day of temperature, speech became thick, much less intelligible, though deglutition was not then interfered with, the patient taking and retaining large quantities of liquid nourishment. Mental hebetude had given place to restless, low delirium, with subsultus tendinum and occasional risus sardonicus. The patient was seen at nine P. M. on this day, by Dr. Landon Carter Gray in consultation with the writer, and the diagnosis of tubercular meningitis confirmed. Slight epistaxis occurred on the fifth day, and the patient passed urine involuntarily for the first time.

The absence of cremasteric and abdominal reflexes was noticed on the same day, as well as the presence of taches cerebrales. From this time the urine was passed through catheter, the quantity being enormous, and sugar being present by analysis. The condition of the patient remained about the same until the evening of Sept. 17th, three days before death, when speech became almost unintelligible, hebetude or stupor greater, and deglutition very difficult and imperfect. The pulse ran up from 80 to 140 in a few hours, respiration becoming also affected. Considering these symptoms an indication of serious involvement of the

¹ Ophthalmoscopic examination of the fundus revealed negative results.

vagus, death was hourly expected. All food and medicine had to be discontinued by mouth. To combat the impending failure of heart and respiration, $\frac{1}{25}$ gr. strychnine was administered hypodermically, and repeated in three hours. These injections were continued at irregular intervals, and with decided effect in stimulating both respiration and pulse. Two days before death it was noticed that the patient, in his restless and ataxic movements of the extremities, scarcely used the right leg or arm at all, and this condition gradually developed into a distinct right hemiparesis.

On the morning of the 19th there occurred a discharge of thick muco-purulent offensive matter from the nostrils, which continued up to within an hour or two of death. It escaped with each expiratory movement, and the amount was estimated to have been at least four or five ounces. Toward the close the temperature and pulse rose rapidly—the former registering, half hour before the end, 106° in the axilla. Death occurred quietly. There was no convulsion at any time, nor was there the slightest tendency manifested in that direction.

I fortunately secured an autopsy, which was made, ten hours after death, by my friend Dr. E. G. Mason, assisted by the writer. The contents of the skull alone were examined, in accordance with a promise previously exacted. Upon removing the scalp, a cup-shaped excavation in the centre of the external surface of the right parietal bone was observed. It was about as large in circumference as a silver dime, and was the result of a local necrotic process, probably tubercular. It did not extend to the inner table, and there was not the slightest pathological change in the corresponding subjacent meninges. There was also a depressed linear fracture (quite old) of the outer table of the right frontal bone, about half an inch above the orbital ridge. Although at first there appeared to be some atrophy of the convolutions corresponding in location to this fracture, it was afterward determined that this appearance was the result of a temporary derangement in handling of the hemispheres. The bones of the skull were unusually thin. Superficially examined before removal, the dura was seen

to be studded at irregular intervals, along the convexity of both hemispheres, with granular masses, the surrounding meningeal tissue being intensely congested for a limited area. These small masses, upon section, were cheesy in consistency, and were evidently pathological in character. They were differentiated from the Pachionian bodies, and were presumably tubercular. Upon removal, the brain (which I show you to-night) was found to be normal in size and in the development of the convolutions. It weighed fifty ounces. The dura was adherent only at two or three places, principally at the base, where the essential pathological conditions were found, so far as the pathology of the case has been investigated. It will have been noticed that the specimen presented is that of a perfectly intact brain, no sections having been made. This was unavoidable in order to show the characteristic appearance of the basal ganglia, which would have been impossible had the brain been dissected. The condition observed at the base of the brain, upon removal, corresponded to a lepto-meningitis involving the upper medulla and pons, the crura cerebri, and all the cranial nerves perhaps. The location of the area of disease had been easily determined by the symptoms observed during life, and the correspondence was exact in location, but by no means in degree. The area described was found covered with purulent exudation, perhaps two ounces in amount. This was of viscous consistency, and clung tenaciously to the surface of the brain. The brain-tissue itself was in a state of beginning disintegration or necrosis, and the sheaths of the nerve-trunks were filled with pus. The pia-arachnoid was intensely injected in places and in others softened and easily torn. No tubercles, clearly demonstrable as such, could be found in the pia-arachnoid at any point.

The case, to summarize it briefly, offers more than one inviting field for speculation and inquiry. Considering it first from an etiological standpoint, it would be interesting to know the exact relationship of the patient's temperament, his mental surroundings, and accumulated sources of anxiety and brain worry, to the development of tubercles in the meninges. What relationship, if any, existed between

the apparently stationary condition of the pulmonary disease to its cerebral development? In other words, are we to expect, in patients affected with tuberculosis of a similar temperament and surroundings, the development of the cerebral disease, and cannot such development be prevented by a proper recognition of this fact, if fact it be, with the enforcement of brain rest? Coming next to the clinical aspects of the case, could an earlier diagnosis have been made? Obstinate insomnia and headaches, it will be remembered, were the only subjective symptoms manifested until within two weeks of a fatal termination. Would it not, however, have been perfectly justifiable to have diagnosed cerebral tuberculosis with these two symptoms alone, knowing that the patient suffered from the pulmonary disease? Viewing the case from a therapeutic standpoint, the "old-new" question suggests itself: Has the surgeon a place in the treatment of such cases? Here was pus, and a surgical axiom demands that pus be evacuated wherever found. The skull has been entered so often for this purpose that it is now an old story. If we are to consider the spontaneous escape of pus, which occurred in this case, a cue from nature, the question is already answered in the affirmative, and the surgeon has but to imitate.

The most interesting features of the whole case, however, are to be found in the results of the autopsy, the most remarkable being the gross disparity between the very grave and extensive lesions found post-mortem and the comparative mildness (although the patient died) of the corresponding paralytic symptoms observed during life. Six or eight ounces of offensive pus (including the amount discharged through the nares and the ounce or two found at the autopsy) would, one would think, have provoked symptoms of a much more pronounced character, involving by pressure, as it did, such an extensive area and structures of such vital importance and sensitiveness. The only solution of the enigma is to be found in the oft-repeated statement, that the brain, in spite of theories to the contrary, is capable of resisting, to an enormous and unknown degree, the encroachments of disease or injury of even the gravest character.

A NEW ELECTROPION.

BY CHARLES HENRY BROWN, M.D.

THE medical use of electricity is much hampered by its many inconveniences. This is seen especially in the use of elements which have for their excitant the "red acid" fluid. In spite, however, of its destructive character and uncleanness, and for the want of something better, it is in universal use at the present time. It is a compound of one part sulphuric acid, fifteen parts water, and one part bichromate of potash. When a physician views the havoc made by this preparation on his clothes, carpets, or buggy robes, he may well exclaim, remembering the cause, as did Spartacus remembering his wrongs: "I shall not forget it! But should memory fail, there are scars here to quicken it!" The portable battery has in many instances been replaced by stationary cells on account of this preparation which annoyed and troubled its owner. The acid fumes emitted, as well as the creeping salts, render the "red acid" element difficult to keep in good working order. Zincs are eaten away, carbons become encrusted, and repeated cleaning and realmagamating necessary with the renewing of zincs, makes this form of battery a nuisance. To those who have any experience with the "red acid" fluid it is not necessary to dilate upon any of its disagreeable features.

There would certainly be a hearty welcome given to any excitant that is cleanly, non-corrosive, and gives as strong a current.

This heretofore has not been accomplished, though attempts have been made over and over again.

My attention has recently been called to a new improvement in battery fluids, invented by Mr. Henry C. Beers, of this city, named by him "The Monogram Battery Fluid."

It is a strong solution (7 per cent.) of the sulphate of mercury, containing a trace of nitric acid, prepared in a special manner, its exact composition and mode of preparation being at present withheld for commercial reasons. It is a colorless, odorless fluid, of a high electro-motive force (1.7 volts), will not polarize, gives a strong and constant current, will not discolor the skin, and is harmless as to staining or destroying any fabrics with which it may come in contact. The zincs are kept automatically amalgamated, and a much greater quantity of current can be taken from a cell with a far less consumption of zinc than with the "red acid" fluid. Its electro-motive force is less to commence with than that of the "red acid" cell, but it is more constant. The rapidity with which the "red acid" cell runs down, soon makes them equal, the "monogram fluid" maintaining a long period of mean constancy after the "red acid" fluid has become comparatively useless. Should the elements be accidentally left in the fluid, the chemical action is so slight that the destructive action on the zinc is nil.

The following comparative tests were made by me between the "red acid" fluid and the "monogram fluid" on the 13th of October, 1890.

One cell, of the size ordinarily used in portable galvanic batteries, was used, containing two ounces of the fluid in each case. The elements were one each of zinc and carbon, three and one-eighth inches long, one inch wide, and three-sixteenths of an inch thick, separated from each other a distance of one-quarter of an inch. A Waite & Bartlett milliampere-meter, the same one for each test, was used; the resistance-box and conducting cords were also the same, so that the conditions and surroundings were in all respects alike in each test.

In the following table, the first column gives the time that elapsed after the immersion of the elements, an observation being taken every half hour for five consecutive hours. The second column gives the reading of the milli-ampere-meter at the time of taking the observations, for the "monogram fluid," and the third column the same for

the "red acid" fluid, one test following immediately after the other. Each zinc was carefully weighed before and after the test in which it was used. The "red acid" fluid was composed of one part sulphuric acid, fifteen parts water, and one part bichromate of potassium. Total resistance in the circuit, 28 ohms.

<i>Time elapsed after immersion of zincs.</i>	<i>"Monogram." Current in milliamperes.</i>	<i>"Red Acid." Current in milliamperes.</i>
$\frac{1}{2}$ hour	50	66
1 "	50	62
$1\frac{1}{2}$ "	50	53
2 "	49	50
$2\frac{1}{2}$ "	48	43
3 "	47	30
$3\frac{1}{2}$ "	46	20
4 "	45	15
$4\frac{1}{2}$ "	30	11
5 "	24	10
Total	439	360

<i>"Monogram Fluid,"—Zinc Element.</i>	<i>Oz.</i>	<i>Dr.</i>	<i>Sc.</i>	<i>Gr.</i>
Weight at commencement of test	2	0	1	18
Weight at termination of test	2	0	1	7
Loss in zinc in grains	11			

<i>"Red Acid Fluid"—Zinc Element.</i>	<i>Oz.</i>	<i>Dr.</i>	<i>Sc.</i>	<i>Gr.</i>
Weight at commencement of test	2	0	3	15
Weight at termination of test	2	0	1	15
Loss of zinc in scruples (or 40 grains)	2 0			

Total quantity of current—"Monogram Fluid"	439 m. a.
" " " " "Red Acid Fluid"	360 "

Extra quantity in favor of the "Monogram Fluid"

Percentage greater in favor of the "Monogram Fluid"

Total percentage of quantity of current derived in favor of the "Monogram Fluid," taking into consideration the comparative consumption of zinc: "Red Acid Fluid," 40 grains; "Monogram Fluid," 11 grains

It will be seen by the above figures that the current given by the "monogram fluid," although not so forcible to commence with, yet, taking all things into consideration, is

more constant and greater in force and quantity for a given length of time than that generated by the "red acid" fluid. The "monogram fluid" in five hours' time loses but little less than half of its strength, while the "red acid" loses a little more than seven-eighths, with a consumption of nearly four times as much zinc, or as 11 is to 40.

Considering the observation taken at the end of the fifth hour, it will be seen that the "monogram fluid" could have continued its work much longer, or as 24 is to 10, than could the "red acid" fluid.

The new fluid possesses the quality of cleanliness while it is devoid of the destructive and disagreeable characteristics of the "red acid" fluid.

CEREBRAL LOCALIZATION.

Ratenof (*Bolnechnia Gazeta Bolkina*, i., 1 and 2, Feb. 7, 1890) reports the case of a student, twenty-two years of age, who accidentally shot himself with a revolver. The ball, about eight millimetres in diameter, entered the posterior parietal region on the right side. The immediate effect, aside from the shock, was a complete loss of sight. There were no decided motor or sensory symptoms: the patient complained only of his blindness. The eyes seemed entirely natural, and the ordinary reflexes to light, etc., were normal. There was some general disturbance, fever, etc.; and the second day after the injury the wound was opened and probed to the depth of four or five centimeters, and the clot of blood that had gathered removed, and vision returned, with left lateral hemianopsia.

The patient survived the injury, with various symptoms of headache, epileptiform attacks, etc., six months, and the autopsy revealed softening of a triangular section on the external surface of the right hemisphere, extending backward from the point of entrance of the bullet near the posterior extremity of the Sylvian fissure, and involving the posterior end of the superior parietal gyrus and the upper occipital gyri. On the surface of the left hemisphere the occipital gyri were involved to the apex of the lobe. The description of the internal faces of the hemispheres is not clear, but the cuneus must have been implicated in both hemispheres. The bullet, somewhat altered in shape, was found in the left hemisphere.

Periscope.

By LOUISE FISKE-BRYSON.

TUMOR OF THE CORPUS CALLOSUM.

The "American Journal of Medical Sciences," June, 1890, contains Henry J. Berkley's observations upon this rare condition. Gowers and Seeligmüller are the only recent writers who have touched upon the subject. There are, however, scattered through different journals, twelve cases recorded by Mills (1), Glæser (3), Maguire (1), Bristowe (4), and Bruns (3). There are present in this condition no pathognomonic symptoms that can actually refer to the corpus. Headache and mental dulness are the most frequent, but no inference can be drawn from them. Optic neuritis is rather important, existing in about a third of the cases. Other symptoms are hemi-paresis, paraparesis, muscular stiffness, vomiting, and epileptiform attacks.

INSOMNIA AND ITS TREATMENT.

This is the title of an admirable editorial in the "Medical and Surgical Reporter" of March 22, 1890. The wise physician at once gives up the thought of routine treatment when it is a question of insomnia, giving to etiology and therapy most careful consideration. The pressing need for this has elicited contributions from many able writers, notably two recent papers—that of Jastrowitz in the "Berliner klinische Wochenschrift," July 3, 1889, and that of Krafft-Ebing in the "Wiener klinische Wochenschrift," 1890. The causes of sleeplessness are in many instances difficult to determine. As yet, the causes of sleep are not known. The mechanical theory is the most probable, that sleep is produced by anæmia of the brain; but this is by no means certain. Cerebral anæmia may be the result of sleep, not its cause. A dynamic theory has been advanced, according to which sleep results from the resting of portions of the brain that preside over consciousness and volition. Preyer's chemical theory accounts for sleep on the ground that tired muscle produces some toxic substance that acts upon the brain.

The most frequent cause, probably, of obstinate insomnia is brain overwork and nerve overstrain, whether through work or worry. Still more powerful than overwork is the

exercise of the emotions, of whatever nature. While the mind should be at rest, it is instead entertaining multitudinous sensations of fear, hope, anger, revenge, and the like. If these causes persist, insomnia becomes habitual. Closely allied to this kind of sleeplessness is that depending on actual mental and nervous disease. Great fatigue is also a cause of wakefulness, as are fevers, anæmia, malnutrition, *mania à potu*, and coffee, tea, and tobacco. The insomnia of uræmia, often an early symptom of Bright's, may be included with last mentioned four forms as toxic insomnia. Inability to sleep may be a secondary condition, as when pain, cough, palpitation, polyuria and thirst prevent normal rest. A frequent cause—if it can be styled a cause—is old age. With this extended group of diverse etiological factors, proper treatment is seen to be a matter of greatest difficulty. The acting cause must, of course, be removed at once. Work, theatre-going, thrilling romances, the entertaining of company, the use of tea, coffee, and tobacco, must be forbidden as evening indulgences. The application of water is often useful, and bodily exercise before retiring. Certain individuals are unable to sleep when reclining, but drop asleep readily when sitting up. While feasting often murders sleep, the sensation of an empty stomach is by no means conducive to repose. A glass of hot milk and a cracker or two half an hour before going to bed may often help to woo tired nature's sweet restorer.

Another requisite is that sleep should be solicited. The patient must spend a quiet evening, go to bed in a cold and darkened room, exclude company, relax the muscles by proper position, and make a firm and persistent effort to go to sleep, regardless of any impulse to move, whatever its cause. Great effort is often necessary to follow this injunction. Whether it is that the intense concentration of mind on the one purpose acts as does hypnotism is not known, but the result is that sleep will often come surprisingly soon. The feet should not be cold, as this greatly interferes with sleep. Massage, electricity, and hypnotism are distinctly useful. In excessive bodily or mental fatigue, Lauder Brunton gives strychnine at night, in the effort to bring the nervous system from the conditions of over-fatigue to simple fatigue, and thus induce sleep.

First in the list of drugs that produce sleep stands opium and its preparations. It is, however, rather a narcotic than a hypnotic, and is not to be recommended in most cases. Chloral is still the most powerful hypnotic

and is rarely called for. Chloralamid now attracts great attention. It is certainly useful, and less apparently dangerous than chloral. As its action on the heart and respiration is not yet positively determined, it must be used with caution. Paraldehyde, amyline hydrate, sulphonal, hyosine and cannabis indica (see "British Medical Journal") suggest themselves as useful hypnotics.

A GROUPING OF THE VARIOUS FORMS OF INSANITY.

In M. Allen Starr's book, "Familiar Forms of Nervous Disease," Frederick Peterson has a chapter on insanity, in which occurs the following simple and natural grouping of mental alienation that serves most admirably the requirements of student and general practitioner:

I. Defect of Brain—

- (a) Idiocy.
- (b) Imbecility.
- (c) Feeble-mindedness.
- (d) Psychical degeneracy (paranoia).

II. Diseases of Brain—

- (a) Mental depression (melancholia).
- (b) Mental exaltation (mania).
- (c) Mental enfeeblement (dementia).
- (d) General paresis (dementia paralytica).

RESTRICTION IN THE VISUAL FIELD IN SYRINGO-MYELIA.

"Médecine Moderne," July 17, 1890, notes the fact that Déjerine and his pupil, Tuilant, have begun some researches upon this subject, and have found changes in the field of vision in five cases out of seven. In each case the field for green is the most restricted. In vary degree, and not permanently, the other zones present abnormalities.

PSYCHOSES FOLLOWING INFUENZA.

In the "Annales Medico-Psychologiques," July, 1890, Ladame, of Geneva, presents some interesting facts upon this subject. The febrile diseases in which most frequently are observed psychoses of convalescence, or asthenic psychoses, are acute articular rheumatism, pneumonia, variola, intermittent fever, typhoid fever, scarlatina; and exceptionally, measles and pertussis. Psychoses following influenza were scarcely recognized prior to the recent pandemic, though Bonnet, of Bordeaux, observed a case of violent mania following grippe in 1837, and Crichton-Browne cites a case of acute dementia after influenza in 1874. Deli-

rium may usher in grippe, as it does other acute infectious diseases. Ewald, of Berlin, gives the following striking example: A child of seven went to school in the morning apparently perfectly well. Instead of returning home, he boarded a train at the railway station, saying he wished to go to Leipsig. The conductor put him off, the child being unable to tell his name, age, or address. Through the activity of the police, he was soon restored to his parents, whom he failed to recognize. For several days violent delirium continued, which proved to be the precursor of grippe. The prominent psychoses due to influenza are melancholia, sometimes with suicidal or homicidal tendencies, hypochondria, hypochondriacal melancholia with delirium (*depressives Wahnsinn of Krapelin*), and four forms of rapid decay of the mental faculties depending upon sudden exhaustion, viz., delirium of collapse, hallucinatory dementia, asthenic dementia, and acute dementia or stupidity. These are all primary conditions, denoting a state of brain senility. The victims, whatever their age, have grown old under the influence of grippe. Physicians in Vienna consider *nonna* or *nona* an absence of reaction from the general prostration that grippe causes. It is an asthenic psychosis terminating in lethargy and coma, which attacks preferably persons worn out with work or worry who have been unable to care for themselves properly during their attack of grippe. Depression of spirits, great irritability, and dislike or indifference to those around—a condition resembling the mental state of neurasthenia—are other nervous manifestations. Organic insanity may develop during or following influenza in persons predisposed. But the poison of this disease is not the cause, only the last straw. Prognosis in the post-grippled psychoses is good. Except in grave forms, they disappear in a few days, a few weeks or months, or possibly last a year. Treatment consists in restoring the general tone, and combating symptoms as they arise. For insomnia, prolonged warm baths, and the wet pack, followed by a light collation of some warm dish, with wine or tea; as sedatives, moderate doses of bromide of potassium, and sulfonal at night; for great excitement, hypodermics of hyoscine. The presence of sitiophobia is an indication for artificial feeding.

INSANITY PROCEEDING FROM THE COLON.

The earliest writer to call attention to the colon as a reflex cause of insanity was Schroeder van der Kolk. It formed no mean division of his order of sympathetic insan-

ities. An article on the subject by Harold Moyer appeared in the "Alienist and Neurologist," January, 1890, in which the author suggests that in these days of germs and ptomaines, the theory of auto-infection will be more acceptable to many. Whether as an irritation beginning in the peripheral endings of the sympathetic nerves, propagated to the vaso-motor supply of the central nervous system and there working disorder principally in the circulation, or as an addition to the blood of noxious elements that produce toxic effects, the disorder manifests itself by an intellectual disturbance, characterized by a peculiar depression of spirit, by anguish of mind, and by the patient's self-accusations of wickedness and baseness. The disease has a very slow course and generally this anguish has existed some time before a physician is consulted. Van der Kolk was himself a sufferer, to the degree of hallucinations and phantasms that appeared for three days. Copious enemata that removed enormous quantities of fecal matter cured him; and Dr. Moyer's patients—three cases of typical insanity—were restored by similar measures. Drastics only increase the tendency to stricture. They add to the sensibility of the colon and the accumulation of blood in it, and cause watery stools, while the hard masses in the upper portion of the large intestine still remain. Disquietude and excitement increased, the strength diminished, and the circulation made more irregular by this treatment. Where there is a true over-filling of the bowels, with distension of the pouches of the colon, drastics and cathartics are of little use.

EXCERPTS FROM GERMAN AND FRENCH JOURNALS.

By Drs. F. H. PRITCHARD AND ALBERT PICK (BOSTON).

SCOLIOSES IN SCIATIC NEURALGIAS.

E. Brissaud (*Arch. de Neurol.*, 1890, Vol. 19, No. 55). The deviations of the trunk in sciatica have recently been studied by Charcot, Ballet and Babinsky. There is in many cases of sciatica, according to Babinsky, a more or less pronounced inclination of the vertebral column towards the unaffected side of the body; this symptom is overlooked by many investigators, as it is not especially sought for. The costo-iliac interspace gives one a measurement of the spinal deviation. The shoulder in question is also not always inclined towards the unaffected side, this being prevented by compensatory movements of the spine above the dorso-

lumbar curve, and indeed with over-compensation the shoulder of the unaffected side may be higher than that of the affected side. The head of the patient may be straight, or according to the compensatory curve, it may incline towards one or the other. In diagnosing, one should distinguish between a scoliosis independent of sciatica, a chronic rheumatic process of the sacro-vertebral joints, and hysteric coxalgia.

The cause of the deviation described is muscular, and indeed a simple contraction of the muscles of the unaffected side, resulting from the patient trying to throw the weight of the body upon the sound leg. The leg of the unsound side is easily flexed, and the sole of the foot rests with its entire surface upon the ground.

One sees an example of a professional muscle spasm in the inclination of the shoulder towards the left in violinists.

The atrophy of the muscles on the affected side not rarely extends outside of the region of the ischiadic nerve.

B. then cites three cases in which exceptionally the body was inclined towards the affected side. These cases were spastic, the curvature being caused by a contracture of the muscles, while the "*scoliose croisée*" is only based upon a contracture of the muscles. Not rarely there is an increase of the tendo-reflexes, which, however, may be seen in every painful affection of the lower extremities. Not rarely the "*ischiasis*" is not a neuralgia in the region of the sciatic nerve, but of the lumbo-sacral plexus; from these various contractions and contractures of the loin- and leg-muscles the different deviations in the position of the legs and body originate.

ON THE NERVOUS FORMS OF THE GRIPPE.

Prof. L. Revilliod (*Rev. médic. de la Suisse Romande*, 1890, No. 3). R. gives an interesting contribution to the knowledge of the nervous forms of the grippe, adding it to several cases chosen from those under his observation. The most prominent place is given to delirium, which he also most fully describes.

The intensity of the delirium stands in a misrelation to the objective symptoms which often are scarcely remarked. It nearly always accompanies the pneumonia of influenza, but, however, is to be closely distinguished from the usual pneumonia delirium, for in pneumonia the delirium sets in at the stadium acmes, and in fatal cases persists until death; but in the grippe it is often the harbinger of pneumonia, or it may appear in the stadium decrementi and last

long after the pneumonia. Delirium may also appear without pneumonia; it is often an accompanying symptom of a simple bronchitis as well as one of other manifold disturbances of the course of influenza. In some cases they may alternate with other cerebral phenomena.

The sensory delusions during the hallucinatory delirium are usually unconscious, but R. also saw patients who were consciously hallucinatory and who tried by all means to throw them off.

R. also devotes much space to the description of the joint-pains, of which he observed two forms: the neuralgic and rheumatic forms. The latter differ from ordinary rheumatism by the very great hyperæsthesia of the of the skin and bones which this latter never presents. The joint-pains in the grippe are to be placed upon the same plane with those complicating other infectious diseases (pseudo-rheumatism infectieux—Bourey). The disturbances of motility are very variable: paraplegic, spastic, tetanic states, accompanied by bulbar symptoms, produce the most wonderful pictures which are often difficult to recognize; they all had a favorable and rapid course.

But R. also observed a quite malignant form: a male tabic patient and a woman with morbus Basedowii, who had been in the hospital, were attacked by influenza and soon died; presenting the gravest pulmonary symptoms; the necropsy, however, revealed no cause for these.

TREATMENT OF THE UROGENITAL APPARATUS.

Prof. Dr. Moritz Benedikt (Internat. klin. Rundschau, Vienna, 1890). Benedikt first considers enuresis nocturna, which he as a rule regards as easily cured; he employing in its treatment galvanizationi from the bladder outwards, and where this procedure does not succeed the other remedies also will be found to fail. Strangury often remains after diseases of the bladder, especially in hysteric and nervous individuals; it often appears as an independent neurosis (sometimes after defloration or pregnancy).

In the treatment of pollutions he recommends to be taken in the evening, two to two and a half cg. bromium with one cg. each of valerian, zinc, and pulvis folia digit. purpurea. After ten days bromium may be given alone. If this does not succeed, then the psychrophore, cauterization according to Ultzmann, galvanization and faradization through the catheter. Dietetic measures, as the avoiding of eating late in the evening and of alcoholic drinks are also recommended. The therapeutics of spermatorrhœa

are also similar. Three forms of impotence are considered curable :

1. The psychic.
2. The form which appears after great excesses under the form of an irritable weakness, expressing itself by incomplete erections and ejaculations *outer introitum vaginæ*.
3. The paralytic form, seen in individuals with but slight sexual desire and with slight sexual desire or absent ability to have an erection.

Great benefit is obtained from galvanization, electric pencilling and franklinization. As to hydropathic procedures, the employment of the douche (*Fächer-douche*), directed against the back, buttocks, thighs and genitals, deserves attention ; also very cold baths of 7° - 8° , and chalybeate baths.

Suspension, according to him, also plays an important rôle. The psychic influence is also not to be underestimated.

THE ANTI-EPILEPTIC ACTION OF AURUM BROMATUM.

A. Scherscherbak (*Vrach*, 1890, No. 9). The writer's work is based on fourteen experiments made upon dogs, to determine experimentally the action of aurum bromatum upon epileptic attacks produced by stimulation of the psycho-motor region by means of the induction current. The remedy (*Au. Br.*) was given either by the stomach (most frequently), subcutaneously, or intravenously into the femoral vein. After administration of the drug by means of an œsophageal sound, 0.2 g. per kg. body weight, it was no longer possible to provoke epileptic convulsions, in spite of the most violent and continuous stimulation of the cortex. With doses which were two to five times less, epileptic attacks could still be produced, but a greater strength of the current and a longer stimulation were necessary ; but the picture of the epileptic attack was changed, as the convulsion had less inclination to extend, limiting themselves to the side of the body opposite to the hemisphere stimulated.

Nearly the same size of the dose was necessary, subcutaneously. Intravenous injections were remarked to be most efficacious, as 5 mg. pro. kg. were enough to prevent the production of epileptic attacks.

The irritability of the single motor centres was slightly influenced, even from doses of 0.1 to 0.2 pro. kg. The irritability of medullary substance and the latent period of the

muscle twitchings remained with these doses entirely unchanged. Even with such doses it was impossible to provoke attacks from the occipital lobe.

In two cases the animal received in the course of seven days a certain amount of aurum bromatum, and two days after the last dose the influence of accumulated small doses was observed, from which it was seen that a dose which introduced once would produce a slight effect, as, for example, 0.030 pro. kg. perot, after four weeks' administration, would have a distinct and pronounced effect, as no epileptic attacks could be induced. The irritability of the single centres presented no deviations from the normal worthy of remark. There was no action upon the general condition remarked; a decrease of sensibility to pain was only remarked with doses of 0.15-0.20 pro kg.

TETANUS IN GASTRECTASIA.

Dr. M. Loeb (*Deutsches Archiv f. klin. Med.*, Bd. 46) The patient in question was a man, sixty-four years of age, in whom already seven years ago a dilation of the stomach was diagnosed, and, as there was hæmatemesis, an ulcer or carcinoma was thought to be present.

In March, 1889, the patient fell sick with rigors, violent vertigo, frequent and violent vomiting, and pains in the muscles of the calves and jaws. These were followed by painful, tetaniform spasms of the legs and arms; there was reflex rigidity of the pupils (Trousseau's sign) and spasms of the facial muscles. After two hours the attack had passed over. The next day there was a second and shorter attack of tetanus; then general restlessness, increasing confusion of the sensorium, delirium, arrhythmia of the pulse, and cold perspiration. Death on the fourth day of the disease.

Post-mortem examination revealed a high degree of dilation of the stomach, caused by a stenosis of the pylorus, which stenosis was due to a gastric ulcer; emphysema of the lungs; otherwise no pathological, organic changes. The brain was unfortunately not examined.

The writer is of the opinion that the poisoning was caused by the absorption of decomposed substances from the dilated stomach, these acting secondarily with toxic effect upon the central nervous system. Cases of tetanus in gastrectasia have, as a rule, an unfavorable prognosis; out of thirteen cases, collected by the writer from the literature, ten ended fatally.

CLINICAL CONTRIBUTIONS TO OUR KNOWLEDGE OF
TETANUS.

Prof. R. v. Jaksch (Prague, Zeitschr. f. klin. Med., 1890, Bd. 17, Suppl. S., 144). The writer has made a careful study of this disease from very numerous observations which he had at hand. Hence he is able to furnish a valuable contribution to the casuistics of tetanus, which importance is heightened by the observation of many symptoms unobserved up to now. The results of the observations are, in short, as follows:

The majority of the patients, which were males, were mostly seventeen or eighteen years old, and all workmen (nearly fifty per cent. being shoemakers). Most of them fell sick in the months of February, March, and April. Neither heredity nor any preceding diseases were of etiological importance.

General prodromal phenomena precede the beginning of the disease.

The tonic spasms of the extremity always begin at the distal end of the phalanges.

Among the disturbances of sensation, paræsthesiæ are the most frequent and pronounced; trophic disturbances are more rarely observed.

He was able to notice an elevation of the galvanic excitability of the nerves.

No important changes were remarked in the relation of the reflexes.

The average duration of the disease is two to three days. Relapses are very frequent.

What is remarkable is the deviation of the temperature of the body from the normal, which he gives in extensive tables.

The increase in temperature is moderately febrile. In one-seventh of the cases no cause could be discovered; in the other cases intermittent and slight typhoid fever could not be excluded with certainty. Hence the elevation of temperature appears to belong to the picture of tetanus—at least it appears and disappears with the other symptoms, to make way for a subnormal one. Therefore the writer is inclined to accept the probability that tetanus owes its origin to a certain noxa; but unfortunately the attempts of the writer to discover it were negative.

Clinically he calls the disease *acute relapsing tetanus*, and contrasts it with a *chronic* form, which is often observed during pregnancy and after extirpation of goitres.

Finally, tetanus may be the symptom of *grave cerebral disease*, of which the writer gives a striking example.

ON THE PHYSIOLOGY OF THE INFRA-CORTICAL GANGLIA
AND THEIR RELATIONS TO THE EPILEPTIC ATTACK.

Dr. Th. Ziehen (Jena, Arch. f. Psych. u. Nervenkrankh., 1890, Bd. 21). The writer experimented with fifty-three rabbits, proceeding in his experiments as follows: The convexity of the hemispheres is exposed, the dura removed, after double ligation of the longitudinal sinus, and then, with a sharp instrument, avoiding any mechanical displacement, the hemispheres and fornix are cut away, so that the large ganglia become easily accessible.

Touching, puncture, the weak faradic current, and incision were used as irritants. The writer comes to the following results:

1. Mechanical and faradic irritation of the corpus striatum, as well as sections through it, produce sometimes single or tonic twitchings, which, however, do not last beyond the irritation, and are especially prominent upon the opposite side of the body. These twitchings have the character of those which one obtains by irritation of the anterior motor cortical region.

It is remarkable that a nodulus cursorius, in the sense in which Nothnagel used it, was not to be discovered.

The lenticular nucleus of the corpus striatum is not accessible to a direct isolated stimulus; when, however, it was stimulated indirectly, the same results were obtained as from stimulation of the corpus striatum.

2. Mechanical and faradic stimulation of the optic thalamus provoke single, tonic twitchings, which do not persist longer than the stimulus, and were remarked upon the same and opposite side of the body.

Sections in the region of the posterior optic thalami and anterior corpora quadrigemina, involving the base in the region of the chiasma and the anterior pons, produce vehement running movements with locomotion and cries; the region of the tegmentum and the corp. genic. int. are the most sensitive. Mechanical and faradic stimulation of the surface of the anterior corpora quadrigemina are followed by an acceleration of respiration, growling, squeaking, nystagmus, and vehement locomotion.

3. Stimulation of the posterior corpora quadrigemina yields tetanic spasms without locomotion, stronger on the injured side than on the other; these spasms last for minutes after the stimulation has ceased.

The writer explains these effects of stimulation as of reflex origin, and indeed he is inclined to place the path of the running movements with locomotion in the optic tract.

By DR. WM. C. KRAUSS.

In the "*Annales del Circulo Medico Argentino*," for May, 1890, Dr. Alfjandro Castro reports a case of "Tumor Cerebral, Hydatid Cyst of the Left Ventricle, Trepanation, and Death due to Basilar Meningitis." The young man, fourteen years of age, farmer, had always enjoyed the best of health until five months ago, when he began to complain of headaches, followed by vomiting and paresis of the right arm and leg. There exists no hereditary diathesis; the family, consisting of eight brothers, are all living and healthy. He began to complain, five months ago, with headaches, vomiting, etc. These increased in frequency and intensity until October, 1890, when he sought admission to the hospital at Buenos Ayres. His condition at that time is as follows:

Marked atrophy of the right leg, there being a difference of $2\frac{1}{2}$ cm. between the right and left thigh and calf. The difference between right and left arm was 5 mm., between right and left forearm 15 mm. The dorsal interosseous muscles of the foot and the anterior tibial were notably atrophied, as were also the muscles of the hands.

The general sensibility is preserved, although somewhat diminished; reflexes exaggerated; temperature normal; appetite good when he has no headache. There is no deformity of the cranium. Patient refers the pain to the left side of the head.

Although there existed no history of hereditary syphilis, he was administered the iodide of potash for one month without any apparent results. The pains growing more intense, the vomiting more obstinate, and the paresis more marked day by day, it was resolved to resort to operative procedure.

After carefully following all antiseptic measures, the trephine was applied over the fissure of Rolando. Through a small opening in the meninges a blackish, soft substance, hernia-like, made its appearance. Enlarging the aperture in the cranium to 65.40 mm., a blackish tumor, the size of a walnut, covered by the meninges—congested, but non-adherent—made its appearance. It was easily removed with a curette, and, after carefully ligaturing all meningeal vessels, a drainage-tube was inserted and the wound dressed. The first six days passed without any noteworthy events. The headaches, vomiting, and the paresis seemed to have notably diminished, appetite improved, the wound was cicatrized and healthy, and the only discharge from the tube was some arachnoid fluid and some softened particles of brain-matter.

On the seventh day, December 26th, the discharge was slight, but there was a bulging outward of the meninges. On the following day it had increased to the size of a hen's egg; and on the third day it was the size of an orange. On the 31st of December the tube was removed and a weak solution of boric acid was injected into the wound, when suddenly the cicatrix gave way and a large cyst, 7 cm. in diameter, was ejected. It contained 200 grammes of a clear yellowish liquid with some flakes, which fell to the bottom of the vessel. The membranes of the cyst were easily separated, and were about 2 mm. in thickness. On the inner membrane there existed numerous whitish granulations, which, under the microscope, proved to be echinococcus cysts adherent to the germinating membrane. They were vesiculated, full of living echinococci. The day after the expulsion of the cyst the temperature fell to 40°, pulse 120.

On January 2d, temperature 41°, pulse 130. On dressing the wound another cyst, the size of a hen's egg, made its appearance and was removed. An epidemic of diphtheria was reigning at the hospital at this time, and the wound was covered with false membranes. The condition of the patient grew worse until January 13th, when he died, basilar meningitis being given as the *causa mortis*.

In the June number of the same journal, Dr. Maglioni, of Buenos Ayres, describes a case of a young man, who, having never suffered from any nervous affection, fell, striking his head against a nail, and soon afterward was seized with epileptic convulsions. The wound was located over the (superior-posterior) *cephalo-dorsal* portion of the parietal bone.

He is very taciturn, complains of headaches, cardiac palpitations, no paralysis, no disturbance of the general sensibility. The attacks—which are truly epileptic—are general, not localized, and occur sometimes every day, sometimes several times during the day. On trephining, no fracture or depression was found, dura not congested, no abnormal condition of the underlying brain-substance. The wound was dressed antiseptically and healed nicely.

Five weeks afterward, March 15th, occurred the first attack; March 17th he had three attacks; April 28th, two attacks; and April 30th, one attack. From that time, up to reporting the case, May 20th, the patient has been free from any epileptic seizures. The writer wishes to draw no conclusions from this case, but feels compelled to say that, in cases of traumatic epilepsy, trephining is justifiable.

In "Revista de Ciencias Medicas de Barcelona," June, 1890, Dr. Robert describes an interesting case, which he designates as a "filhelio," or "sun-lover." The case is that of a boy, just at the age of puberty, strong and vigorous, showing no deformity or sign of any retarded development, living in the mountains of Cataluna. There is a slight neuropathic history in his family, and at times he is under the influence of alcohol. For the past ten years he has exhibited a strong desire for the sunlight, looking for hours at the sun without the least inconvenience. At times he takes an attitude—legs separated, hands clinched, head turned backward, eyes fixed on the sun—and remains in this position until exhausted, then becomes unconscious and falls to the ground; he is seized with convulsions, tonic and clonic, and after a short interval awakens. The writer regards the case as epilepsy with an unusual sensorial aura.

By LOUISE FISKE-BRYSON.

POST-DIPHTHERITIC RESPIRATORY PARALYSIS CAUSING
PULMONARY COMPLICATIONS.

In the "American Journal of American Sciences" (September, 1890) there is a paper upon this subject by W. Pasteur, of London, who thinks that too little attention has been paid to the effects upon chest-movements and affections of the thoracic viscera following post-diphtheritic paralysis. While cardiac paralysis is well recognized, the influence of diaphragmatic or intercostal paralysis upon the lungs, and through them upon the pulmonary circulation and the heart, has been strangely overlooked. The classical signs of diaphragmatic paralysis are: Reversal of respiratory movements of the epigastrium and hypochondria; dyspnœa on exertion or excitement; diminished force of cough, sneezing, spitting, etc.; and loss of compressive action of the abdominal muscles, difficulty of defecation, etc. (von Ziemssen).

The more constant and obtrusive physical signs presented by the cases presented in the paper were: 1. Increased movement of the lower ribs, observed in ten out of fourteen cases; not noted in three. 2. Altered movements at the epigastrium during respiration in twelve out of fourteen cases. 3. Altered character of cough and voice; noted in ten cases, but probably present in all.

Tube-feeding was necessary. Paralysis of the lower limbs was the rule, and in some the arms were affected;

also in a few the muscles of the back and neck were involved. Knee-jerk was absent in every case. The facts brought forward by the author leads him to the conclusion that paralysis of the diaphragm or other parts of the chest-wall tends to induce loss of function in the subjacent lung, which is, *cæteris paribus*, proportionate to the degree of paralysis, and results in more or less *collapse and œdema of pulmonary tissue*. This state of affairs renders it imperative to guard in every possible way against the occurrence of broncho-pneumonia.

When swallowing is followed by attacks of coughing, feeding by means of a soft rubber catheter, introduced through the nose, gives an opportunity for sufficient nourishment and prevents the leakage of food into the air-passages. To assist chest-expansion to the utmost, artificial respiration (Sylvester) should be practised on the first appearance of any indication of diaphragmatic paralysis. It is not so much the treatment of asphyxia that is important as that of the condition which gives rise to it. In hospitals this treatment is carried out by the nurses three or four times in twenty-four hours, for a period of ten or fifteen minutes, instruction being given to disturb the patients as little as possible and to keep them well covered.

CHLORALAMID (SCHERING)

is now one of the favorite hypnotics. It is only about a year since its introduction, with competition firmly fixed upon the market and among the armamentarium of the physician.

There are numerous data to its value, and the agents for its sale in this country have gathered these from all sources, in pamphlet form. The various experiences and facts presented are instructive, and, as they assert, no doubt will convince the skeptical as to its being a harmless and reliable hypnotic.

Chemically.—It is a chloral-formamid, discovered by Prof. J. von Mering. It occurs in the form of colorless, slightly bitter crystals, melting at 239° F. It is soluble in from nine to fourteen parts of cold and less of warm water, in one part of absolute and one and a half parts of ninety-six per cent. alcohol. It requires five hours to dissolve twenty grains in two ounces of water, and only fifteen minutes when the menstruum is one dram of rectified spirit. No precipitation occurs on adding the alcoholic solution to

water. Locally, chloralamid has been found to be absolutely free from irritation, even when applied to the conjunctiva.

Chloralamid induces an apparently natural sleep in from one half to three hours. The only unpleasant effects that have been noted are occasionally headache, lassitude, and a desire to sleep in the morning. The best results are obtained when insomnia is due to neurasthenia, hysteria, old age, and to such conditions as chronic alcoholism, cardiac and bronchial asthma, subacute nephritis, diabetes, and other chronic conditions. Aulde considers its chief advantages lie in the fact that it is not objectionable to the palate, and that its effects are soon manifested. Brainerd calls attention to the fact that it is more soluble and less expensive than sulfonal. The danger of a large dose is less than in chloral. Hagen and Huefler, of Erlangen, pronounce it one of the most reliable hypnotics.

Administration of Chloralamid.—Much depends upon the proper administration of the new hypnotic, chloralamid, to obtain the full effect and satisfactory and beneficial results. The dose is from 15 to 60 grains, with an average dose of 30 grains. Chloralamid is soluble in about 20 parts of cold water, and in one and a half parts of alcohol.

An additional caution is necessary: *Never dissolve or dispense chloralamid in hot water or warm solutions*, as the heated preparation decomposes.

The best modes of administration are:

1. In a teaspoonful of whiskey or brandy.
2. In properly proportioned solutions with wine, spirits, or spirituous compounds.
3. In a small cup of cold water or cold tea.
4. In powder form, in wafers or cachets washed down with cold water.

The following formulas have come well recommended and bear the stamp of general approval and adoption:

Dr. W. Hale White (in "British Medical Journal") says: "I always prescribe it with spirit; 20 grains will dissolve in 1 drachm of rectified spirit in fifteen minutes, and water may be added to this solution without reprecipitating the drug. A good way of giving it is to tell the patient to dissolve it in a little brandy, add water to his liking, and drink it shortly before going to bed."

Asylum Notes.

At a special meeting of the State Commission in Lunacy, held at the Capitol, in the City of Albany, on June 1, 1890, the following was ordered :

1. (a) That the Superintendent or officer in charge of each institution for the care and treatment of the insane be directed not to permit the service of any legal process whatever upon any insane patient except upon the order of a judge of a court of record, which shows that the judge had notice of the fact that the person sought to be served was at the date of the order an inmate of such an institution.

That at the time the service of any process is made the following proceedings must be had :

The nature of the process, the date of the same, name of the court out of which it issued, and the date of its service must be entered in the history of the patient in the case-book.

The order upon which the service is made and the process must be filed with the papers relating to the patient.

A copy of the process, together with an explanatory letter, must be forwarded at once to the committee of the person and property of the patient, if there be one, or, if there be no committee, then to the nearest known relative or next friend of such patient.

(b) That no insane person be permitted to sign any bill, check, draft, or other evidence of indebtedness, or to execute any contract, deed, mortgage, or other legal conveyance, except upon the order of a judge of a court of record, which shows that the judge had notice of the fact that the person whose signature is sought to be obtained was at the date of the order an inmate of an institution for the care and treatment of the insane.

That at the time of the execution of the order the following proceedings must be had :

The Medical Superintendent, one of his assistants, or officer in charge must be present at the time of the execution of the order and must see that its terms are strictly complied with.

The substance of the order and the proceedings had thereunder must be entered in the history of the patient in the case book.

The order must be filed, with the papers relating to the patient, and a copy of the same, together with a notice of the proceedings had thereunder, must be forwarded at once to the committee of the person and property of the patient, if there be one, or, if there be no committee, then to the nearest known relative or next friend of the patient.

2. That the Medical Superintendent or officer in charge be directed to keep a copy of these orders posted conspicuously in the general reception-room and office of each institution for the care and treatment of the insane.

At a special session of the State Commission in Lunacy, held at the Capitol, in the City of Albany, on the second day of September, 1890.

In the Matter of the Admission of Private Patients to the State Hospitals for the Insane.

It appearing that large numbers of the insane poor are deprived of the benefits of the intended care and treatment which the State Hospitals were instituted to provide for them; that much space at these institutions, originally provided for the accommodation of that class, is now occupied by private patients; that the law known as the State Care Act reaffirms the policy of the State and declares the insane poor to be the wards of the State, and provides for the removal as rapidly as possible of those now remaining in the county poor-houses to the State Hospitals: therefore be it

Ordered—1. That on and after October 1, 1890, no private patient at any State Hospital be permitted to occupy more than one room for his or her personal use or behoof, or to command the exclusive services of an attendant; and, thereafter, there shall be no distinction allowed between private and public patients in respect to the scale of care and accommodations furnished them.

2. That on and after October 1, 1890, no private patient be admitted to any State Hospital, except in strict accordance with the statutes, as follows: "Whenever there are vacancies in the asylum" (State Hospital) there may be received "such recent cases as may seek admission under peculiarly afflictive circumstances, or which in his (the Superintendent's) opinion promise speedy recovery," and upon an order granted by the State Commission in Lunacy upon an application in writing, addressed to the Commission, of a near relative, guardian, or committee of the patient.

3. That this order shall not be held, except in special cases, to require the removal of private patients in custody in said hospitals on October 1, 1890.

BOARD FOR THE ESTABLISHMENT OF
STATE INSANE ASYLUM DISTRICTS AND OTHER PURPOSES. }

At a meeting of the Board for the Establishment of State Insane Asylum Districts and Other Purposes, held at the Capitol, in the City of Albany, Tuesday, September 2, 1890.

In accordance with the provisions of Section 1 of Chapter 129 of the Laws of 1890, the following division of the State into State Insane Asylum Districts was made and ordered to take effect October 1, 1890:

Utica State Hospital District—Counties of Albany, Fulton, Hamilton, Herkimer, Madison, Montgomery, Oneida, Saratoga, Schenectady, containing 1,476 insane patients.

Willard State Hospital District—Counties of Allegany, Cayuga, Chemung, Livingston, Ontario, Schuyler, Seneca, Steuben, Tompkins, Wayne, Yates, containing 1,024 insane patients.

Hudson River State Hospital District—Counties of Columbia, Dutchess, Putnam, Rensselaer, Washington, Westchester, containing 1,159 insane patients.

Middletown State Hospital District—Counties of Orange, Queens, Richmond, Rockland, Suffolk, Sullivan, Ulster, containing 988 insane patients.

Buffalo State Hospital District—Counties of Cattaraugus, Chautauqua, Erie, Genesee, Niagara, Orleans, Wyoming, containing 1,148 insane patients.

Binghamton State Hospital District—Counties of Broome, Chemung, Cortland, Delaware, Greene, Otsego, Schoharie, Tioga, containing 548 insane patients.

St. Lawrence State Hospital District—Counties of Clinton, Essex, Franklin, Jefferson, Lewis, Onondaga, Oswego, St. Lawrence, Warren, containing 964 insane patients.

The number of public insane patients in each of the above-named districts is given as it exists on this date.

At a special session of the State Commission in Lunacy, held at the Capitol, in the City of Albany, on the second day of September, 1890.

In the Matter of the Charge to the Counties of the State for the Care and Maintenance of Insane Patients.

The Commission having before it estimates and special reports from the Superintendents of the several State Hospitals and other interested persons concerning the charge to be made to counties of the State for the care and maintenance of insane patients in the said hospitals, and said Commission being required by statute to establish a charge for maintenance, which shall be the same for all the counties of the State, it is

Ordered—1. There shall be charged for each patient in continuous custody under the commitment, or order, by which he is held, as follows:

(a) For the first three years or less, the sum of four dollars and twenty-five cents per week.

(b) For any period of time exceeding three years, the sum of two dollars and fifty cents per week.

2. The charge hereby established shall include food, clothing, breakage, and all other charges of any name or nature, and no greater charge shall be made under any circumstances whatsoever.

3. This order shall be in full force and effect on and after October 1, 1890, and shall apply to all patients in custody on that date.

In the Matter of the Transfer of Public Insane Patients from their Homes or from Poor-houses to State Hospitals by Superintendents of the Poor.

The statute (Section 6 of Chapter 126 of the Laws of 1890) having made it the duty of the President of the State Commission in Lunacy to prescribe regulations governing the transfer of public insane patients from their homes or from poor-houses to State Hospitals by Superintendents of the Poor, and concerning the clothing of said patients, it is on this 10th day of September, 1890, hereby

Ordered:—1. That all County Superintendents of the Poor or town, county or city authorities, before sending a patient to any State Hospital see that said patient is in a state of bodily cleanliness and provided with the following clothing, to wit:

(a.) One full suit of underclothing.

(b.) One full suit of outer clothing, including head wear, boots or shoes.

Between the months of November and April, both inclusive, there shall be provided, in addition to the foregoing, a sui able overcoat for the men patients and a suitable shawl or cloak for the women patients; also gloves or mittens. Considering the great, of the introduction of contagious or infectious diseases into institutions where large numbers of people are congregated, and to avoid, so far as possible, the introduction of such diseases by means of wearing apparel, the clothing above provided for must in all cases be new.

2. In traveling by rail patients must not be compelled to ride in smoking or baggage cars, except in the case of men patients who may be so violent, profane or obscene as to render their presence in ordinary passenger coaches offensive. If any portion of the route is necessary to be traversed by team, a covered conveyance should, unless impossible, be provided. The shortest practicable route should be selected; the hour of departure should be timed, so far as possible, so as to avoid the necessity of stopping over night on the journey and so as not to reach the hospital at an unseasonable hour. Whenever practicable, a notice in advance, by writing or telegraph, should be sent to the Medical Superintendent of the Hospital of the coming of the patient. In cases of violent patients a sufficient number of attendants should be provided to control their actions without resorting to the use of mechanical restraints, such as straps, ropes, chains, hand-cuffs, etc.; quieting medicines should not be given to such patients except upon the prescription of a physician. If it becomes necessary to remain over night or for a number of hours at a station on the route, patients are not to be taken to jail, police station or lock-up. Food in proper quantity and quality, and at intervals not exceeding five hours, should be provided for patients, but no alcoholic beverages must be given unless upon prescription of a physician. Opportunity must be afforded for attention to the calls of nature, and the rules of decency must be observed. In case of the employment of extra attendants in conveying violent patients, care must be taken that they are of adult

age and of good moral character. The provisions of the statute which require that a woman attendant shall accompany women patients when taken to State Hospitals must be strictly complied with.

3. Any violation of the requirements of this order shall be promptly reported, so far as known to him, by the Medical Superintendent of the Hospital to the State Commission in Lunacy.

4. This order shall take effect on the 1st day of October, 1890.

By the President of the Commission :

T. E. McGARR,

[L. S.]

Secretary.

Dr. Paul Gibier, Director of the New York Pasteur Institute, sends us the following report :

Since its opening (February 18, 1890), to date, 610 persons, who had been bitten by dogs or cats, came to be treated. These patients may be divided in two categories :

1st. For 480 of these persons it was demonstrated that the animals which attacked them were not mad. Consequently the patients were sent back after having had their wounds attended, during the proper length of time, when it was necessary. *400 patients of this series were consulted or treated gratis.*

2d. In 130 cases the antihydrophobic treatment was applied, hydrophobia having been demonstrated by veterinary examination of the animals which inflicted bites or by the inoculation in the laboratory, and in many cases by the death of some other persons or animals bitten by the same dogs. *ALL THESE PERSONS ARE TO-DAY ENJOYING GOOD HEALTH. In 80 cases the patients received the treatment free of charge.*

The persons treated were :

64 from New York.	1 from Maryland.
3. " New Jersey.	1 " Maine.
12 " Massachusetts.	1 " Kentucky.
8 " Connecticut.	1 " Ohio.
9 " Illinois.	1 " Arizona.
3 " Missouri.	1 " Iowa.
3 " North Carolina..	1 " Nebraska.
3 " Pennsylvania.	1 " Arkansas.
2 " New Hampshire..	1 " Louisiana.
2 " Georgia.	1 " Ontario (Can.)
2 " Texas.	

Dr. E. H. Van Deusen, formerly and for many years superintendent of the Michigan Insane Hospital, has just presented to the city of Kalamazoo the sum of fifty thousand dollars for the purpose of erecting a public library building, with the proviso that said building shall be set aside one of its rooms for the exclusive use of the Kalamazoo Academy of Music.

Stanley's recent Emin Expedition was equipped with Fairchild's Digestive Ferment, and in the recent attack of gastritis from which Mr. Stanley suffered, he was entirely sustained upon foods previously digested with Fairchild's Extractum Pancreatis.

A HYGIENIC GAS BURNER.

A wholesome atmosphere in the sick-room is one of the most important remedial agents which should engage the attention of every physician. Many physical disorders occur as a natural sequence of antedating influences. The physician's calling is dual in character: one to prevent disease, the other to cure it; one being as much a part of his duty as the other. In this double capacity we find exercise for both governing principles of practice when dealing with the chemistry of air. Their first duty is to prevent atmospheric vitiation, and if such occurs, in spite of our precept and effort, then it becomes our secondary and next duty to rid ourselves of the objectionable condition by specific and adequate ventilation or by neutralizing deodorizers and disinfectants.

An ordinary individual would hardly expect to find a hygienic agent in a gas burner, nevertheless such we have found to be the undubitable truth.

Illuminating gas consumed in any burner abstracts oxygen, the life-sustaining element, from the atmosphere, and substitutes the products of combustion. As combustion is more or less complete, so is the deterioration of the air. By present methods of burning gas only about one-half the possible light of a cubic foot is utilized, the other half passing into the air as poisonous products of imperfect combustion. When ordinary gas is properly consumed, the vitiation of the air is insignificant, but when consumed improperly, the deadly and obnoxious carbonic oxide (an exclusive product of imperfect combustion) makes its appearance, and no one can follow its stealthy and insidious effect upon all who dwell and sleep within its pernicious influence. We require but one thing to mitigate the evil, a burner which will at all times, no matter how the pressure varies, consume the gas properly, an ever watchful guard against imperfect combustion and the making of carbonic oxide, a little automatic barrier betwixt life and death, a machine missionary to keep in action and perpetually add to the comfort of mankind. Combined with a perfect combustion of gas are several valuable features which are harmonious joint issues with the action, such as steadiness of flame, uniformity of illumination, and economy.

We have practically tested the Jackson Automatic Regulating Gas Burner, and pronounce it perfectly constructed, beautifully finished, untiring in its operation, doing all that is claimed for it, and should be used in the sick-room, home, church, store, place of amusement, hospital, public school, or wherever gas is required. The Jackson Cold Drawn Steel Co., located at 21 Jane Street, manufactures it.

We heartily endorse the efforts and success of this Company in placing this sanitary burner economically upon the market, and we heartily commend it to the attention of our profession.—E.D.

Society Reports.

AMERICAN NEUROLOGICAL ASSOCIATION.

*Sixteenth Annual Meeting, held at Philadelphia, June 4th,
5th, and 6th, 1890.*

(CONCLUDED.)

Dr. BURT G. WILDER, of Ithaca, N. Y., exhibited the left hemisphere of Chauncey Wright, and made the following remarks:

Mr. Wright was a well-known philosophical writer and general critic, who died in Boston in 1875, at the age of forty-five. His brain was removed by Dr. Thomas Dwight, now professor of anatomy in the Harvard Medical School, who published a brief description of it, with a diagrammatic figure of the left side, in a paper entitled "Remarks upon the Brain of a Distinguished Man" ("American Academy Proceedings," 1877, xiii., 210-215). It has since been generously loaned to the writer by Prof. Dwight for fuller study and publication.

A large number of photographs of the two hemispheres have been taken, from all aspects, oblique as well as direct, and copies of these photographs are herewith submitted. In the "Reference Handbook of the Medical Sciences," viii., the lateral aspect of the left hemisphere is shown in Fig. 4779, and in Fig. 4781 the dorsal aspect of the entire cerebrum with the ends of the occipital fissures and the central fissures, which latter are completely interrupted by an isthmus at about the junction of the middle and dorsal thirds. The present exhibition is mainly for the sake of enabling members of the Association to see an example of this rare fissural anomaly, the number of cases observed hitherto not exceeding fourteen out of the thousands of brains examined. It seems probable, however, as remarked by Heschl and Dwight, that there is not infrequently an incomplete interruption of the central fissure, constituting a *radum* (shallow, or "concealed *pli de passage*"), and this should be looked for when practicable.

About fifteen points of interest respecting this brain are noted in the "Handbook," p. 158. Pending a future complete description, the following additions or corrections are made now:

The semicircular fissure just ventra of the postcentral is probably the *subcentral*, first observed by the writer in brains numbered 385 and 2268, in the museum of Cornell University ("Handbook," viii., 152, note). The partial exposure of the insula in this brain of a highly intellectual person is especially noteworthy in connection with its similar exposure in an uneducated mulatto, and its complete occlusion in most apes and monkeys. A careful study of the insula will be made by the removal of the several operculums, kindly authorized by Prof. Dwight.

In the published figure the bifurcated fissure between the orbital and the ventral end of the precentral is identified as the presylvian ("ascending branch"). Recent studies of this region have thrown doubts upon this determination and upon Herve's affirmation as to the greater constancy of the subsylvian ("horizontal branch") in Primates.

In this connection the attention of members of the Association was asked to the printed "Commentary upon Fissural Diagrams;"¹ also to the series of large wall-maps (about two metres square), recently made under his immediate direction, by Mrs. S. H. Gage, from specimens prepared by him for Cornell University. They embrace: (*a*) the mesal aspect of the entire head of a man and a chimpanzee whose brains were hardened in the cranium by continuous alinjection; (*b*) the lateral aspect of the brain of the chimpanzee and an adult mulatto; (*c*) the brain of a child at birth; (*d*) six human foetal brains at as many stages of development, illustrating especially the formation of the Sylvian fissure.

THE CONTRACTION OF THE HEART AND ORDINARY STRIATED MUSCLE,

was the title of a paper, presented by Dr. Thomas J. Mays. It is well known that under certain conditions the degree of contraction of a skeleton muscle varies with the stimulus applied to it. Inquiries into the nature of cardiac contraction (Bowditch, Kronecker, Stirling) have taught us that the heart differs widely in its mode of contraction from that of striated or skeleton muscle. The feeblest stimulus which is capable of calling forth a contraction acts like the strongest—the most complete or maximum contraction being, therefore, induced by the weakest or minimum stimulus (Landois). The cause of this behavior is supposed to be inherent in the structure of the heart muscle itself. The

¹ Copies may be had upon application to Dr. Burt G. Wilder, Ithaca, N. Y.

muscular elements of the heart partake of the function of nerve fibres (Aubert), and on account of the anatomical variations the heart differs in its function from that of other striated muscles.

The experiments of Wundt and Walton show that during strychnine poisoning "a stimulus which is strong enough to produce any reflex contraction in a muscle will not react more strongly if the greatest possible stimulus is applied. The range of stimuli through which the contraction varies with the intensity of the stimulus becomes shorter as the effect of the poison increases, and when a certain grade of poisoning is reached the step is infinitesimal from a stimulus which produces no contraction to one which produces a maximum." (Walton.)

When irritability is viewed, therefore, as it exists normally in voluntary muscle, it is seen that varying degrees of contraction are obtained from this organ when varying degrees of electric stimulation are applied to it; but this differential responsiveness vanishes so soon as its irritability is increased through the influence of strychnine. The irritability of the normal heart he regarded as being analogous to that of voluntary muscle under the influence of toxic doses of strychnine, for here, in virtue of the heightened irritability of the heart, the degree of electric stimulation which provokes a cardiac contraction at all is too powerful to produce anything else than a maximum contraction.

DIPHTHERIA AND DIPHTHERITIC PARALYSIS.

was the title of a paper, read by Dr. James Hendrie Lloyd. The weight of opinion seems to be that the specific poison of diphtheria is either a bacterium or the ptomaine generated by it. This gives us at least a working theory, upon which an active and even aggressive treatment can be established. Diphtheritic poison acts distinctly like other morbid agents upon nerve tissue. There is little doubt that it acts especially upon what Gowers calls the lower "segment" of the nervous system; that is, the large cells in the anterior horns and the nerve fibres running out from them to form the nerve trunks. That some observers have located the lesions in the horns and others in the nerve fibres only, furnishes additional evidence that these two parts form really one anatomical organ, and that the diphtheritic poison acts probably upon the whole. It might even be claimed on good clinical evidence that it is a general protoplasmic poison which does not confine itself to one group of cells. Arsenic, alcohol, and some other substances attack many tissues of

the body and are similar in their effects to the diphtheritic poison. If a violent protoplasmic poison is being generated in the body, the most important indication is to prevent its generation by early local treatment. Secondly, having the lesions of a peripheral neuritis, it is possible to give an intelligent prognosis, and to carry out efficient though somewhat expectant treatment.

Chlorate of potassium seems to be losing the confidence of the profession in the treatment of diphtheria, because large doses have an injurious effect upon the kidneys. Chloride of ammonium will do all that chlorate of potassium can do, without its bad effects. Calomel is a good remedy, without the same prompt results as the combined chlorides of ammonium and iron. The corrosive chloride of mercury, both locally and internally, appears on some accounts the ideal remedy for diphtheria.

Nervous phenomena must not be regarded as sequelæ of diphtheria, but as a part of the general symptom group. Bernhart pointed out that the knee-jerk is abolished in many cases of diphtheria which do not exhibit distinctly paralytic symptoms. But the broader fact is that a toxic agent begins early and probably in all cases, to threaten the integrity of the peripheral nervous system. Heart failure, due probably to involvement of the vagus, is the most alarming symptom, and occurs without other paralytic manifestations. In general multiple neuritis of diphtheritic origin, the prominent symptoms may not be noticed for several weeks after the disappearance of the primary disease. The treatment of the paralysis itself is in the main expectant. Nerve lesions demonstrated in autopsies are degenerative—destructive. There is destruction of the axillary fibre, segmentation of the medullary substance, and proliferation of the cell-elements in the nerve-sheath. The repair must be by a gradual and rather slow nutritive process.

Strychnine has been recommended, and digitalis in heart failure. Alcohol, as a more diffusible stimulant, is better than digitalis. The most important indication is to feed; or, as has been said, "to keep the blood-vessels and lymph spaces full." The phosphates, especially in the old form of Parrish, are invaluable in the treatment of diphtheritic paralysis.

In adults, diphtheritic paralysis can be mistaken for locomotor ataxia, as in the case of a man who had numbness of the extremities, slight ataxia, abolished patellar reflexes, and suspicious change in the pupils. The principal points

of distinction were the loss of muscular power, flabby muscles, absence of fulgorant, absence of Romberg's symptom and the history of earlier diphtheria. The changes in the pupils in diphtheritic paralysis are the opposite of those occurring in the Argyle-Robertson phenomenon of locomotor ataxia. In diphtheritic paralysis the power of accommodation is lost to near objects, and not the reflex to light.

Dr. KNAPP was much surprised at the statements made by Dr. Lloyd as to the treatment. That diphtheria was due to a localized infection might be granted. It was, however, of as little use to attack local manifestations of hysteria or the initial lesion of syphilis, in the hope of getting rid of those troubles by that means. Certainly Dr. Lloyd's method of using a long-handled brush down the patient's throat seemed very remarkable, and, for young children, absolutely impossible or likely to do more harm than good. The use of syringes, irrigation by the atomizers and with steam, laden with disinfectants, would certainly not irritate or excite the patients, and seemed infinitely better treatment. If there were marked constitutional symptoms, every effort must be made to keep up the strength. The speaker always used strychnine in large doses, and in almost all cases it had been well borne. The chief danger, apart from the heart failure, which was not as late a manifestation as the true diphtheritic paralysis seemed to be failure in nutrition from paralysis of the pharynx. This was a point which should be early guarded against.

Dr. LLOYD said that modern research showed that we had a local infectious disease to deal with, from which point the system became involved, and we must attack the disease locally. The brush was to be preferred to the spray. The latter was useful for the nasal chambers. Chloride of iron and ammonia should be used for the throat. He did not advocate caustic applications, but only mild antiseptics, lotions thoroughly applied to the local disease.

TUMOR OF THE QUADRIGEMINAL REGION, WITH SPECIAL REFERENCE TO OCULAR SYMPTOMS.

Dr. B. SACHS read a paper with this title. He had been fortunate enough to obtain two autopsies, during the past year, which bore upon this question, and also several cases which were subjected to careful clinical examination. His first case was one of unusually severe tuberculosis cerebri. The main points of the history, which he had been able to complete through the kindness of several colleagues, were these:

E. L——, aged three years. When first seen she had double ptosis, but no other ocular paralysis was observed. She was dull and listless, and had a pulse that ranged from 145 to 160, but with normal temperature. The mother had noticed a change in the child's disposition since an attack of measles nine months previously. The child did not care to play, but preferred to sit quietly in a chair all day long. She staggered in walking and occasionally fell. She had no epileptic attacks, and had vomited but once. Knee-jerk was absent. The right hand was weaker than the left. No anæsthesia or ataxia. There was paresis of both levator palpebrarum, the pupils being half covered. No nystagmus. Pupils were equal, moderately dilated, and reacted well to light and accommodation.

December 28, 1889, the patient had come under Dr. Sachs' care. The condition at that time showed great changes. Examination disclosed double and almost complete ptosis. There was no upward or downward movement of either eye. Both external recti muscles were thrown into clonic spastic condition when the attempt was made to use them. The interni were capable of very slight movement, but all the other ocular muscles were completely paralyzed. The accommodative reflexes were still distinct, and there was slight contractility to light. There was also slight left facial paresis. The vision was very much impaired. Although in a semi-stupor, the child could be made to walk, and then exhibited most distinct cerebellar staggering, walking with a broad base, and almost falling to the right side. The oculist reported plaques of choroidal atrophy below the macula of left eye. The reflexes were exaggerated, and there was occipital headache. The diagnosis of tumor of the corpora quadrigemina was given. The tumor was supposed to be associated with a general tubercular meningitis. The child grew rapidly worse, and, after passing through several convulsive seizures, becoming blind, and finally developing left hemiplegia, she died February 4, 1889.

Autopsy showed the dura adherent to the skull, and it had to be removed with the calvarium. The quantity of the sub-dural fluid was slightly increased. A solitary tubercle was at once discovered near the right lateral sinus, pressing into the lateral edge of the cerebellum and producing thrombosis of the lateral sinus. Other tubercles with large areas of softened tissue were found in the cerebellum. Deep examination of the brain showed the hemispheres to be healthy, with the exception of the small

tubercular deposits along the paths of the blood-vessels. The cerebellum was the seat of the most profound changes. The base presented several unusual conditions. There was great thickening of the pia, with small tubercular deposits between the corpora mamillaria and optic chiasm, and in the interpeduncular space. The thickening at this point was so great that both third nerves, instead of lying across the crura, after removal of the brain, pointed backward, and the right, the sixth, was twisted out of its position.

Section of the brain showed the tumor to occupy almost the centre of the tegmental division of the crus, and had left a very small portion of the corpora quadrigemina and the brachi intact. The occipital headaches and the cerebellar staggering were the only symptoms which could be ascribed to the large tubercles in the cerebellum, though both these symptoms might be due to the lesion of the quadrigemina region. It was probable that the sixth and seventh nerve-nuclei were responsible for the symptoms pointing to lesions of those nerves, or that the basilar meningitis was at fault. Certain it was that the latter condition was late in developing, for, for months, the symptoms had been distinctly nuclear. In spite of the manifold morbid conditions, it was most remarkable that the ciliary muscles and the sphincter irides had remained exempt during the entire period of observation. Considering the compactness of all cerebral structures in the crura, it would be supposed that there could be no difficulty in making a differential diagnosis between cases of tumor in this region and a chronic inflammatory process.

CRUS LESION.

This was the title of a second paper by Dr. SACHS. Crus lesions were rarer than many other cerebral lesions, but their symptoms were well marked. The case under consideration had some special interest, however, in connection with post-hemiplegic disturbances of motion, and from this point of view the results of the post-mortem examination were worthy of consideration.

Seven years ago the patient, a woman, about fifty years of age, had had a dizzy attack one morning, and had found her vision rather blurred. There was a recurrence of the attack in fifteen minutes. There was no unconsciousness nor difficulty with speech, but when the patient attempted to walk she found she could not with ease. By morning she had almost complete left hemiplegia; she could not open either eye. At that time speech was heavy and indistinct, but from this she had recovered in three weeks.

Hearing, taste, and smell were altogether normal. The hemiplegia was never recovered from, the patient became somewhat unruly and demented, and was finally taken to the Montefiore Home, where she had remained for many years.

A few further details of the patient's chronic condition were elicited in examination. There had been no history of syphilis, but there was very marked atheroma of the peripheral arteries. In addition to the left hemiplegia, the patient had suffered amputation of the right leg above the ankle, for old necrosis of the tibia, fully six years before. There was rigidity of the left leg, and increased knee-jerks of both sides. The wrist reflex was decidedly increased on the paralyzed side, but the left upper extremity was subject to the wildest ataxic movements. This would go on until the arm dropped from exhaustion, when it would remain quiet until aroused again by an effort to use the hand. She became extremely emotional, took very little nourishment, and finally died. The diagnosis of crus lesion of the right side, probably softening from thrombosis, was made, and confirmed by the autopsy.

NEW YORK NEUROLOGICAL SOCIETY.

Meeting of October 7th, 1890.

The PRESIDENT, Dr. L. C. GRAY, in the chair.

Dr. W. B. PRITCHARD presented a specimen, and the history of a case of "Tuberculous Meningitis," see p. 720.

CAN WE DIAGNOSE HYPERÆMIA OR ANÆMIA OF THE BRAIN AND CORD?

Dr. WILLIAM A. HAMMOND read a paper on this subject. The writer had for many years been familiar with a group of symptoms, which, from their etiology and general characteristics were indicative of cerebral disturbance, and some twenty-five years ago, after considerable observation and many experiments performed upon living animals and the human subject, he had come to the conclusion that they were the result of an increase in the amount of blood circulating in the vessels of the brain. His conclusions were first published in an article on "Insomnia," in 1865, various papers appearing on the subject at subsequent intervals, and lastly in a monograph issued in 1884, entitled "Cerebral Hyperæmia the Result of Over Mental Work or Emotional Disturbance," in which additional facts, the outcome

of continued experience were brought forward in support of the theory advanced. In the writer's opinion there were certain symptoms which positively indicated the existence of cerebral hyperæmia, and which he had designated as symptoms of the first class. There were others which inferentially led to the same conclusion, especially when they were associated with symptoms of the first class. Those were embraced under the term symptoms of the second class. Others again were indicative of derangements of various organs of the body, which, though important as adding to the discomfort of the patient, might be due to many different primary pathological states and therefore were not included in the present discussion. The symptoms of the first class were, first, wakefulness; second, pain, heat, a feeling of fullness or distention in the head, a sensation of a band encircling it, a dragging and clawing sensation at the vertex, vertigo and hallucinations, provided, and this point was especially impressed, that these symptoms were increased by any known factor which increased the amount of blood in the brain. Third, a congested condition of the tympanum, and the optic disc, the retina, and the choroid. The theory which the writer had advanced repeatedly was that natural sleep was due to a comparative anæmic condition of the brain, normal wakefulness to an increase of the amount of blood in the cerebral vessels, and insomnia to an abnormal quantity of intra-cranial blood. Persistent insomnia was the necessary accompaniment of the pathognomonic symptom of the affection in question. Without wakefulness there was no cerebral hyperæmia; with cerebral hyperæmia there was always wakefulness. Numerous experiments made upon animals, had fully demonstrated those facts. It was well-known that during the process of digestion there was a diminished amount of blood in the brain, and it was for this reason that persons felt sleepy after ingestion of a hearty meal. Although those observations and experiments were conclusive enough, further demonstration had been made by means of an instrument devised for the purpose of determining the existence of cerebral hyperæmia. By its means observations were made upon the movements of the brain and the blood-pressure within the cranium. It consisted of a brass tube, which was screwed into a round hole made in the skull with a trephine. Both ends of this tube were open, but into the upper was screwed another brass tube, the lower end of which was closed by a piece of very thin sheet india rubber, and the upper end with a brass cap, into which was fastened

a glass tube. This minor arrangement contained colored water, and to the glass tube a scale was affixed. This second brass tube was screwed into the first, till the thin rubber pressed upon the dura mata and the level of the colored water stood at O, which was in the middle of the scale. Now, when the animal went to sleep, the liquid fell in the tube, showing that the cerebral pressure had been diminished, an event which could only take place in consequence of a reduction in the quantity of blood circulating in the brain. As soon as the animal awoke the liquid rose at once. The experiments were performed upon dogs and rabbits, and in every instance the pressure was lessened during sleep and increased during wakefulness. The writer thought that nothing could exceed the conclusiveness of experiments of this character. Of the second group of symptoms, hallucinations being the most remarkable, was the only one considered. A number of cases were cited from the recorded experience of the writer and other observers. In most of the cures reported, the spectre or apparition had appeared to the persons on retiring to rest, or on inclining forward, and vanishing when the erect posture was assumed. The explanation of such cures was very simple. The recumbent position facilitated the flow of blood to the brain, and at the same time tended, in a measure, to retard its exit. Hence, the appearances were due to the resulting congestion. As soon as the individuals rose in bed, or stood erect, the reverse condition existed, the congestion disappeared, and the apparitions went with it. Hallucinations of hearing were not infrequently produced by like causes. A number of cases were related to illustrate this point. The writer did not want to be understood as saying that there was a fixed condition of the fundus of the eye and the tympanum, which was associated with cerebral hyperæmia, but that observations should be made from day to day in each case, when it would be found that as the other symptoms of cerebral hyperæmia disappeared, the retina, the choroid, and the tympanum would lose their congested appearance. So that when health was restored the fundus of the eye, and the drum head were found to be very different from what they were when the disease was at its height. There were certain agents, which by their action appeared to increase the amount of blood in the brain, and others, which apparently diminished it, and which were hence important in their diagnostic relations. If to a person suffering from insomnia, pain in the head, vertigo, and hallucinations, should be given one or two hundredths

of a drop of nitroglycerine, the trouble would become unbearable. Like effects followed the use, at such a time, of quinine, strychnine, and other agents. Among those remedies used to diminish the amount of blood in the brain, the bromides stood preeminent. Another diagnostic fact, was in the action of ergot. As was well-known this substance possessed the property of constricting the organic muscular fibre. The writer was convinced from personal investigations that ergot did contract the cerebral vessels, and hence it diminished the quantity of intra-cranial blood. The writer said in conclusion that when he had a patient suffering from insomnia, pain in the head, vertigo hallucinations, suffusion of the face, cephalic heat, and other striking symptoms of perhaps less special importance, and when he found these symptoms disappear under the influence of remedies, such as the bromides, ergot, ice and douches of cold water to the nape of the neck, cups in the same locality, nasal blood-letting or spontaneous hæmorrhage, position, and other means calculated to diminish the amount of intra-cranial blood, he did not see how an escape was possible from the conclusion that the patient was suffering from cerebral hyperæmia.

Dr. M. A. STARR said that while he did not wish to be understood as representing those who opposed Dr. Hammond's views, still his convictions at present were those expressed by Dr. Gray in his paper, read recently before the society. The symptoms which had been explained by the existence or assumed existence of cerebral hyperæmia were many of them symptoms which could be produced by other causes. Such, for example, as wakefulness, which was often noticed in individuals when very much exhausted. In puerperal women who had suffered severe hæmorrhage. He had also certainly observed it in patients who were anæmic. Therefore, to say that wakefulness necessarily indicated an hyperæmic brain was to advance a theory which was hardly tenable. Certainly, hyperæmia of the brain might, under certain conditions be diagnosed, but it was a very open question whether this could be done when only wakefulness was present. As to the question of drugs, he had been very much surprised to hear it stated by Dr. A. H. Smith and Dr. Peabody, at a meeting of the Practitioners Society, last winter, that those gentlemen had been treating cases of supposed hyperæmia of the brain with nitro-glycerine and nitrite of amyl. These drugs which were supposed to increase the supply of blood to the brain were being given upon the hypothesis that they dilated the entire ar-

terial system of the body, and the brain would thereby be relieved to a certain extent of blood. The reasoning at least appeared sound. The speaker thought it impossible to base a diagnosis upon any individual symptom.

Dr. J. LEONARD CORNING thought this was not scientific reasoning. The truth might probably be more nearly arrived at by careful induction. If a man came complaining of headache, having a congested face, with a pulse of high tension, whose symptoms could be promptly relieved by pressure upon the carotids, the jugular or bandaging of the legs, might such a patient be assumed as suffering from congestion or anæmia of the brain? The speaker thought congestion. Suppose quinine or alcohol should be given to such a patient and it was found that the symptoms were aggravated it would be certainly concluded that the trouble was congestion.

The PRESIDENT said that of course Dr. Hammond spoke with authority, this they were all prepared to admit. The fact that he was able to do so had much to do with the acceptance of his conclusions without criticism. Still no dictum in relation to a scientific point could be allowed to stand on personal authority alone. The conclusions must bear the force of investigation and be supported by fact. Dr. Hammond must not consider the discussion as having the least personal bearing, but as merely the expression of a general desire to elucidate the problem as far as possible. Dr. Hammond had stated the symptoms of cerebral congestion as being sleeplessness, with a certain feeling of compression or oppression about the head and a flushing of the face.

Dr. HAMMOND here suggested that he had said these symptoms were increased by the dependent posture or by anything which would increase the circulation.

Dr. GRAY accepted the correction and went on to enumerate the conditions in which these symptoms might be found. For instance, insomnia was common enough in mental diseases and worry, melancholia, overwork, constipation and in many conditions in which there was nothing to show that there existed any hyperæmia of the brain. In the early stages of intra-cranial syphilis there was a condition somewhat of the nature of hyperæmia. But then in Bright's disease, in which there was hyperæmia and congestion there existed a condition of stupor. If the list of causes of insomnia were gone through it would be possible to find a certain train of symptoms which would lead to the assumption of existing anæmia in some, and hyperæmia in

others. Experiments had recently been made on the brains of animals, the report of which differed from those of other recorders. As to the point made that the brain rose or increased in volume during the waking period, it was an open question whether this was not due to cellular action producing an increase of blood. As to the association of sleeplessness with the recumbent posture, of course the extended observations of the author of the paper were deserving of due consideration, but so also were the more limited observations of the speaker in this respect, and he had not been able to verify the association. The question before them was not as to the existence of cerebral hyperæmia or anæmia, but as to whether it could be clinically diagnosed. Flushed face might be dependent upon chorea, general paresis or injury to the brain. It was impossible to say whether the symptom was brought on by hyperæmia alone. The feeling of oppression and sense of fulness in the head was found associated with errors of refraction, insufficiency of the ocular muscles, changes of climate, errors of diet, and so forth. To assume that in all those conditions there was hyperæmia of the brain was assuming a good deal, and more than could be proven. It was a point which had not been demonstrated by any pathologist, as to whether there could exist by itself an increased amount of blood in the cellular tissue or other finer structures of the brain without causing disease of the surrounding parts. It was strange that Dr. Hammond after five months' preparation of the subject had cited no autopsies in confirmation of his theory.

Dr. C. L. DANA said he thought it was now generally agreed that there was such a condition as cerebral hyperæmia and that it could be recognized in its acute forms. Such a state might be produced by drugs. Congestive neurosis, trauma, and so forth. The question was and is, what was the condition at the base of that functional disorder which had gone by the name of cerebral neurasthenia; and whether its initial stage was that of hyperæmia or the hyperæmia was a secondary process? An acute and chronic hyperæmia of the brain were conditions admitted to exist, but it was preferable to say functional cerebral neuroses or psychoses, where the hyperæmia was a secondary process, and that seemed the inevitable conclusion to those who watched these cases. Many patients among the neurasthenics showed symptoms of congestion of the brain, others of this class did not in any way present the symptoms of the classic type of cerebral hyperæmia, but showed the condition so shaded down that it was necessary to set aside all the symptoms

generally described. There was something at the back of the hyperæmia. The hyperæmia of the brain was secondary to some disorder of the vaso-motor nerves or to some functional condition involving the whole nervous system. As to insomnia and cerebral hyperæmia, that question was obsolete. To state that sleep was produced by anæmia and wakefulness by the return of the normal amount of blood to the head was, the speaker thought, in the light of modern neurological studies, a theory which could be described as unworthy further investigation.

Dr. HAMMOND thought that his points had been unanswered in the argument. When Dr. Dana said that the neurologists of to-day ignored the theory of the physiological changes during sleep, a theory which the speaker might claim as his own, he thought Dr. Dana in error. He would remind them that he had stated that headache presented innumerable causes for its existence, and it was only when he found it with flushed face, and vertigo, and when it was increased by the dependent position of the head that the diagnosis was certain. Then he knew his patient had hyperæmia of the brain, all the neurologists in the world to the contrary notwithstanding.

THE SENSATION OF ITCHING.

This was the title of a paper by Dr. E. B. BRONSON. He said that it was a somewhat remarkable fact that a manifestation of cutaneous irritability so common as itching, and one with which as a symptom we are so familiar, had been almost entirely neglected as an independent study. Of other anomalies of sensation, such as hyperæsthesia, anæsthesia and pain we had tolerable clear and definite ideas. But what was the cause and nature of pruritis? What this disturbance of sensation? Notwithstanding the fact that the special senses in their present state were so far removed, in respect to the knowledge they yielded to consciousness, from common sensation, there doubtless was a period when the distinction did not exist. Their differentiation had been the result of gradual and long-continued processes of evolution. There could be little question that the sensory organs to which the several senses owed their special attributes had all originally developed from simple nerve endings, that gave but the vaguest intimations of external objects. In this evolution the impelling force, the directing impulse, had been derived from the two grand principles of life known as the instinct of self-preservation and the instinct of reproduction. To one or the other of those instincts every sensation that arose in the body must be referred. All

sensations, as had been shown, were originally tegumentary. To the common integument must be ascribed the source and potentiality of all sensations. As the result of specialization most of those sensations had been withdrawn from the exterior. What traces of the special senses thus abstracted still persisted in the skin might be infinitesimal. There still remained to the skin and adjacent mucous orifices a variety of sensations; others more specialized, including a special sense with perceptive faculties, and finally the most important representative of the reproductive instinct, the aphrodisial sense. The only sense with which the skin was endowed that could be called perceptive and that was worthy of comparison with seeing, hearing, smelling and tasting, was the sense of pselaphesia. It included the sense of contact, which was seen in its most primitive form; its most important element was pressure sense, while the temperature and muscular senses were more or less essential auxiliaries. Common sensation was represented in the integument in its highest positive aspect, by the voluptuous sensations, in its lowest negative aspect, by pain. Returning to the question, what relation to the sensory organs of the skin and to their sensations did the sensation of itching bear? The author believed that there was sufficient evidence to locate the essential seat of pruritus in the epidermis. Itching was evoked by such irritants as acted upon this tissue much more uniformly than by those that acted on the derma. However provoked, the sensation of itching was always associated with a presentment to consciousness as though a foreign body were in contact with the surface. It was that sensation, that experience through many stages of animal life, had taught, was often followed by a prick, or a sting, and the inclination to escape the threatened hurt had grown into an animal instinct. The sense of contact at a minute portion of the sensitive surface was immediately interpreted to mean a miniature attack that must be repelled. If no attack had really been made, but only the threat, then the excitement should disappear without returning the moment the cause producing the sense of contact was withdrawn. But it was this peculiarity of itching that it persisted in spite of such withdrawal, and was only relieved by the act of scratching. It seemed as though the contact, or whatever the change might be that gave rise to the irritation, produced a molecular commotion in the nerves that went on like the jangling of an electric bell, with the continuance of the sensation until such time as the surcharge of nervous energy was released. In pselaphesia the nerve force or the molecular vibrations

excited by the impact was directly transmitted into some intelligent form of activity and the accumulation of nerve excitation, the nervous engorgement, did not occur. The circuit was complete with no point of resistance intervening to obstruction, and so commotion. With regard to the effect of scratching in relieving itching, it was analagous to that produced by muscular exertion, as in those animals in which the platysma myoides was more highly developed than in man, as in the horse and bovine genera, a certain relief might be afforded to pruritic sensation through its energetic contractions, and which was not wholly due to expulsion of the insect or whatever else might have caused the sensation. While some of the phases of itching might be associated with pathological changes in the eperdermis, others had their source more deeply situated and were referable to the nerve centres. To the latter belonged the form of neurosis of which pruritis was at the same time the symptom and sole appellation. Still other sources were doubtless to be found associated with apparently normal physiological conditions. While those represented the most obvious sources of itching or provocations for scratching, there was another factor of which hitherto but little account had been taken. Both the English words itch and itching and the Latin prurio and pruritis, in their secondary significations, conveyed the idea of a longing, teasing desire, while pruritis was commonly used by the Latins as a synonym for lasciviousness. By desire, something more was meant than merely the inclination to brush or scratch away a foreign body of which the sensation was apparently an intimation. It was rather a kind of desire closely akin to a lustful feeling, and one that sometimes made scratching veritably a sensual indulgence. When pruritis reached a certain degree of intensity, the subject was not content with that moderate amount of scratching that would ordinarily create a sufficient diversion to give relief, but there was a disposition to attack the itching surface with a vehemence that amounted to a passion. Recognizing this peculiar element of desire in pruritis, the sexual excitement and depraving tendencies that were so commonly associated with pruritis genitalium was most easily explained. But it was not so surprising that voluptuous sensations should attend itching where they had their natural seat. Such sensations were, however, not confined to the genitalia. They might be concomitants of itching in almost any situation. By means of a violent excitation superinduced by severe scratching, provoked by pruritic irritation, a liberation or discharge of nervous energy took place, accompanied by pleasurable sensations, together with the release of the pruritis irritation. A

temporary inertia and rest followed and continued until a renewal of the pruritis provoked another resort to the same method of relief. As to why these processes were attended with pleasurable sensations, it sufficed to say it satisfied a law of being. Gratification of appetite was a condition of life, either of the preservation of life or of the reproduction of life. The sexual, the aphrodisiac appetite could only be secondary to the instinct and appetites of self-preservation. From the foregoing consideration the following conclusions were drawn :

(1.) That there was a sense of contact independent of the sense of pselaphesia.

(2.) That this sense of contact was the sense disturbed in pruritis.

(3.) That it primarily concerned simple cutaneous nerves, or nerve endings, situated superficially and probably in the epidermis.

(4.) That the disturbance in pruritis was of the nature of a dysæsthesia due to accumulated or obstructed nerve excitation, with imperfect conduction of the generated force into correlated forms of nervous energy.

(5.) That scratching relieved itching by directing the the excitation into four channels of sensation, sometimes, especially when severe, substituting, for the pruritis either painful or voluptuous sensations.

(6.) That the voluptuous sensations might attend pruritis were a manifestation of a generalized aphrodisiac sense, representing a phase of common sensation that had its source in the sense of contact.

Dr. L. D. BULKLEY considered Dr. Bronson's paper one of the most scholarly he had ever listened to. He then referred to some studies he had made as to the reflex character of itching. For instance, if the itching sensation were on the fingers of the right hand, irritation or pinching of that finger would cause a reflex sensation of itching in the neighborhood of the scapula of the same side. He had only found one or two instances in which it was transferred to the opposite side.

Dr. STARR asked whether it was ever thought that itching was a symptom of central nervous disease. Patients with locomotor ataxia were said to be frequently troubled with itching around the arms, scrotum and perineum. He had never seen a case confirming this.

Dr. B. SACHS had never seen it in organic nervous disease, but in functional disorders, such as crural neuralgia, he had known the itching to be more obtrusive than the pain. It was a frequent condition of profound anæmia, and often observed in hysterical women, and in cases of hystero-epilepsy.

Book Reviews.

FAMILIAR FORMS OF NERVOUS DISEASE. By Dr. M. Allen Starr. Pp. 330. Wm. Wood & Co.: New York, 1890.

The title of this volume, it seems to us, is somewhat misleading. The familiar forms of nervous disease, with the exception of epilepsy, chorea, and locomotor ataxia, are indeed very lightly considered. The subject of neurasthenia, which in all of its manifold varieties is probably more frequently observed than any other nervous affection, is condensed into five pages, while other familiar forms of nervous disease, including trigeminal neuralgia, tic convulsif, reflex pains, headaches, their varieties and treatment, fare but little better. On the other hand, what most neurologists consider as *unfamiliar* forms of nervous disease, such, as for instance, circumscribed lesions of the cortex cerebri resulting in Jacksonian epilepsy, and the various monoplegias, hemianopsia, and disorders of the visual tract, the various forms of aphasia and affections of the base of the brain, including hæmorrhages of the pons, thrombosis in the crus, and lesions affecting the various cranial nerves, are dwelt upon with the thoroughness one has a right to expect from an author of Dr. Starr's reputation. They are condensed as compactly as possible, and yet nothing of importance has been omitted. Numerous new and interesting cases illustrate the writer's text and show that the author's experience with rare cases of cerebral disease has been unexceptionably good. But they can hardly be called "familiar forms of nervous disease."

The first half of the volume is devoted to cerebral diseases, and discusses the localization of cerebral functions, the motor area, the visual area, the different varieties of aphasia, subcortical lesions and affections of the base of the brain. These chapters are well written, and although it cannot be claimed that the author advances the standard of the science of neurology by the addition to it of new observations or of new investigations, yet he has summarized and condensed all that is known on these subjects into several entertaining and well-expressed chapters. Dr. Starr is, at times, perhaps a trifle dogmatic. This is particularly noticeable in a few of the diagnoses of rare cases in which an absolute knowledge of the cause and nature of the various lesions is claimed, without, however, being substantiated by post-mortem evidence. This is not a defect of any great moment and detracts but little from the general merit of the work. The latter half of the volume treats of the localization of spinal cord diseases, the paralyzes of infancy, neuritis, paramyoclonus multiplex, epilepsy, and a few functional neuroses. To these are added a chapter on locomotor ataxia by Dr. Winslow W. Skinner; an article on paralysis agitans, and one on the ordinary forms of insanity by Dr. Frederick Peterson; and a few pages on chorea by Dr. Walter Vought. The volume finishes with a chapter on "Electricity as a Therapeutic Agent," and a list of the prescriptions used in the nervous department of the Vanderbilt Clinic.

Dr Starr is to be congratulated on the course he has taken in throwing open to the medical world the statistics and experience derived from the careful observation of so great a number of patients who annually visit the Vanderbilt Clinic, and we earnestly echo the author's hope that this volume may bring forth similar work from other large clinics.

G. H.

INJURIES AND DISEASES OF NERVES AND THEIR SURGICAL TREATMENT.
By Anthony A. Bowley, F.R.C.S. Philadelphia: P. Blakiston, Son & Co.

Contributions to neurological surgery are always welcomed. This valuable one is based upon the author's Jacksonian Prize Essay of 1882, and the Astley Cooper Prize Essay for 1886, with monographs that have appeared in the "Lancet" for 1887 in the form of lectures.

The work throughout bears proof of large clinical experience, and as the author has made careful records and study of these, and brought to bear upon his work logical deductions, it is of unusual value as a guide in the various nerve operations.

The American edition bears the usual impress of great care in preparation that the ever-popular publishers, P. Blakiston, Son & Co., take in all their publications.

THE ANNUAL OF THE UNIVERSAL MEDICAL SCIENCES. 5 volumes.
Edited by Charles E. Sajous, M.D. Philadelphia, 1890: F. A. Davis, Publisher.

This Annual Medical Review, undoubtedly increasing in its influence as well as popularity, so as to have become a positive necessity in the library of every progressive physician, presents this year undoubted signs of great improvement.

That part which is of especial interest to the neurologist is contained in Vol. II., Parts A, B, C, and D.

Dr. Landon Carter Gray has brought to bear his inimitable style in unearthing the progressive researches for the year in the Diseases of the Brain.

Dr. W. R. Birdsall follows in painstaking effort to give the best of the new matter that has appeared on the Various Diseases of the Spinal Cord.

Dr. Henry Hun has ably and completely arranged the most important recent ideas of Peripheral Nervous Diseases, Muscular Dystrophies, and General Neuroses.

No better or more experienced mind and abler pen could have been chosen to cull and write out the kernels of recent advances in Psychiatry. This chapter is by no means to be passed without commendation. Drs. Gray, Birdsall, Hun, and Brush all deserve credit for this analytical and valuable work. Nor could the Editor have better chosen his associates in this field.

Dr. Ambrose L. Ranney, assisted by Dr. Geo. G. Van Schaick, has certainly improved the Department of Electro-Therapeutics, either because there was more that was new to interest in the past

year or more consecutive effort was made in abstracting them in the presenting of the authors' opinions. It is important not to overlook the scientific clinical data in this field of medicine, as it is the only thing of value in placing electricity on a firm foundation as a valuable agent in curing disease, especially in the neurologist's hands, and with whom it seems of late to find less and less favor.

FROM THE STUMP TO THE LIMB is the title of a monograph on the manufacture of artificial limbs, by A. A. Marks, of this city. The various rooms and departments of this extensive establishment form interesting illustrations for the little book. Those interested in this subject are recommended to send to the manufacturer, 701 Broadway, for one of these treatises on artificial limbs.

ELECTRICITY IN THE DISEASES OF WOMEN. By G. Belton Massey, M.D. Second edition. Philadelphia: F. A. Davis.

A new edition of this practical manual attests the utility of its existence and the recognition of its merit. The directions are simple, easy to follow and to put into practice, the ground is well covered, and nothing is assumed, the entire book being the record of experience. So little is known as yet about the pathology of many diseases that are benefited by electricity, and so little about electricity itself, that all records of experience form the most valuable contributions to the study of electro-therapeutics.

FLUSHING AND MORBID BLUSHING: Their Pathology and Treatment. By Henry Campbell, M.D., B.S. (Lond.). London: H. K. Lewis.

A most interesting monograph of over 250 pages. This work is of especial interest to the neurologist, as it considers the "flush of heat" as a complex nerve-storm. It is the only book on the subject since Burgess who wrote in 1824.

It is divided into four parts: 1. Physiological. 2. Flushing. 3. Blushing. 4. Treatment.

The summary is: "Flushing and morbid blushing must not be regarded as by themselves constituting diseases, but rather as manifestations of some defect—temporary or permanent—in the nerve-centres: wherefore our treatment should be directed to the remedy, not simply of the flush or the blush, but of the general nervous defect of which they are the expression."

The careful and complete consideration of all details makes the book a finished one on these interesting vaso-motor symptoms. The physiological section is well illustrated, and the complicated mechanism of the circulation and its relation with the nervous centres is clearly defined.

The reader is promised much that is practically profitable and scientifically suggestive.

FIG. 4. Section through the brain cortex, including the wall of the ventricle from the apex of the occipital lobe.

A. Necrotic membrane containing the majority of the cocci

B. Zone of granulation tissue.

C. White matter.

D. Gray matter.

E. Pia mater showing the exudative meningitis.

The heavily shaded areas indicate the congested blood-vessels.

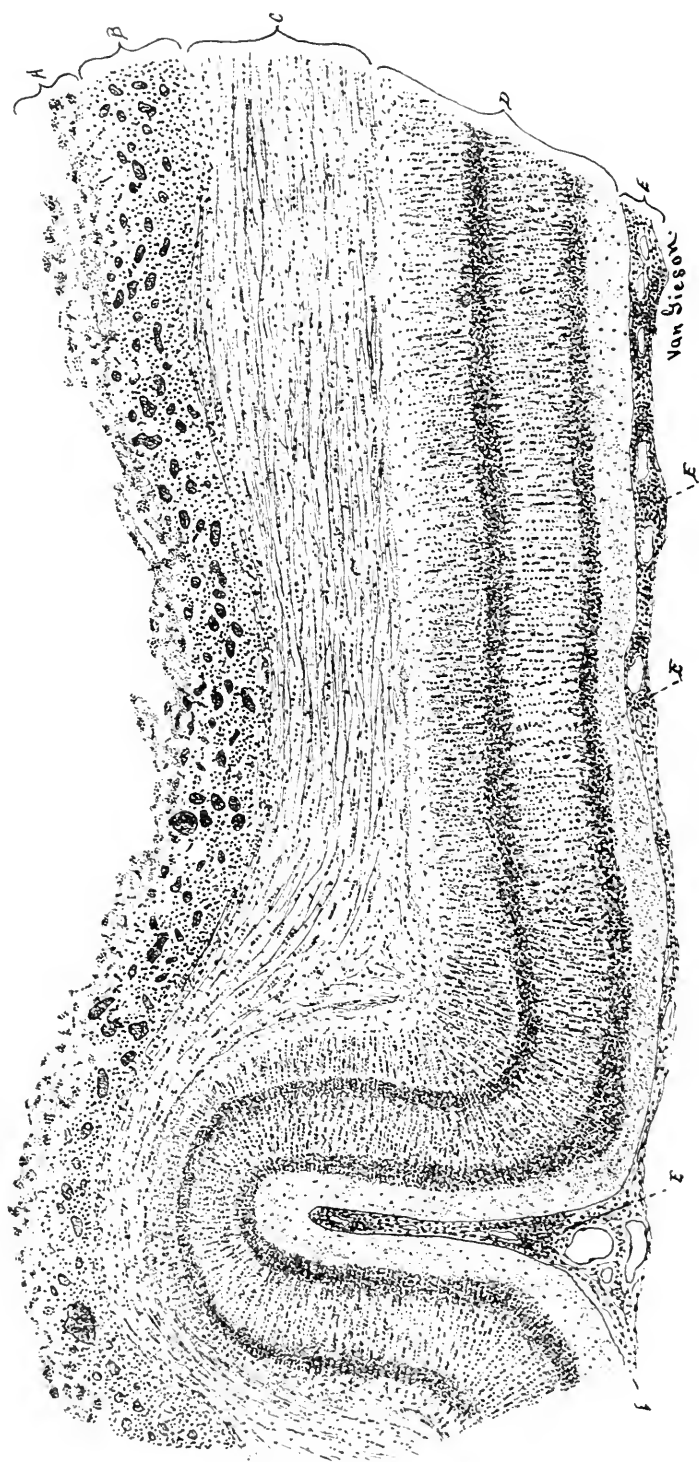


FIG. 3.—Section of the dorsal cord, showing the spinal meningitis, the pellicles on the pia mater, the congestion of the gray matter, the condition of the central canal, and the distribution of the small cocci.

A. Infiltration of the pia mater distending the bottom of the anterior fissure.

B. One of the sulco-commissural vessels passing through the anterior commissure to the margin of the central canal.

C. Canals of the sulco-commissural vessels distended with the meningitis exudation.

V. Spin. ant. and art. spin. ant. anterior spinal vessels. Ven. sept. post. congested vessels of the posterior septum.

Posterior roots.

X. X. Semi-necrotic pellicle on the posterior surface of the pia mater.

FIG. 2.—A section through the wall of the sac, including one of the minute columns of necrotic tissue extending through the thickness of the skin to its outer surface.

a. Zone of exudation and necrotic matter coating the internal surface of the sac containing the small cocci.

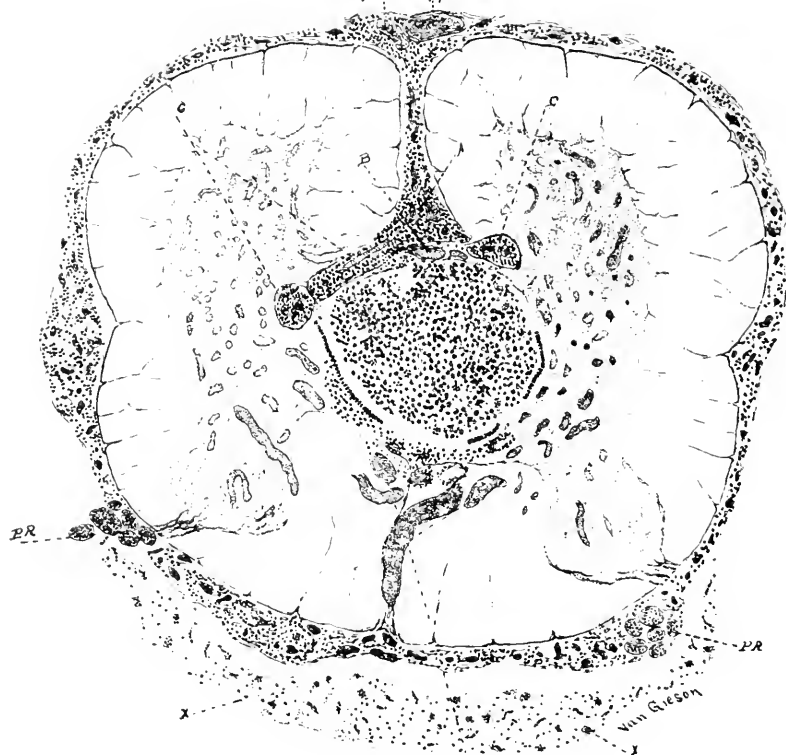
b. Deeper subcutaneous tissue, with congested blood-vessels.

c. Infiltrated corium and papillary derma.

d. Plug-like continuation of the necrosis extending through the skin.

e. Epidermis.

4. Spin. Ant. V. Spin. Ant.



Ven. Sept. Post



THE
Journal
OF
Nervous and Mental Disease.

Original Articles.

A CASE OF SPINA BIFIDA WITH SUPPURATIVE
SPINAL MENINGITIS AND EPENDYMITIS,
DUE TO BACTERIA ENTERING THE
WALL OF THE SAC.¹

By L. EMMETT HOLT, M.D.,
Professor Diseases of Children, New York Polyclinic,

AND

IRA VAN GIESEN, M.D.,
First Assistant at the Laboratory of the Alumni Association of the College of Physicians
and Surgeons.

IN some few cases of spina bifida a suppurative inflammation of the wall of the sac occurs, and leads to a severe and fatal inflammation of the meninges or ventricles of the central nervous system. This complication has attended operations on the sac often enough to be spoken of in the text-books as one of the important contra-indications to punctures or other operations on the wall of the sac. The text-books allude to this complication as purulent arachnitis or purulent meningitis. The percentage of cases of spina bifida in which this complication occurs, or how often the suppurative inflammations of the central nervous system are associated with a like condition of the wall of the sac, is not stated precisely in the available statistics of spina bifida.

The case presented in this paper (which was under the

¹ Read before the New York Neurological Society, Nov. 4, 1890.

charge of Dr. Holt) shows quite clearly how such a purulent inflammation of the wall of the sac and its dependent lesions of the central nervous system are due to the entrance of pyogenic bacteria into the sac, and to their extension along the fluids, meninges, or central canal of the spinal cord, to the brain meninges and ventricles.

The skin of the spinal sac, in spina bifida cases, would seem especially permeable for bacteria. It is frequently extremely thin and the central cicatrix has occasionally a raw exposed surface and lacks the epidermis covering. In surgical operations, pyogenic bacteria have probably been introduced into the sac in the cases where purulent central neural inflammations or suppurative inflammation of sac-wall, or both of these, have followed the operation.

Clinical History (by Dr. Holt).—A female infant, three days old, was admitted to the Babies' Hospital, March 2, 1890. The family history was negative so far as other deformities were concerned. On admission the child was plump and well nourished and presented no physical signs of hydrocephalus, the head being normal in size and the fontanelles not bulging nor tense. There was a lumbo-sacral tumor, measuring $3\frac{3}{4}$ by 6 cm. in diameter and projecting $1\frac{1}{2}$ cm. above the spine. Its walls were exceedingly thin, so that translucency was almost perfect. The skin covering the tumor was healthy except over a central elliptical space—the central cicatrix—directed vertically in the median line, about 1 cm. wide and $2\frac{1}{2}$ cm. long. This extent of the skin, corresponding to the central cicatrix, had the appearance of a granulating surface, and was covered with a sero-purulent discharge. Neither cord nor nerve-filaments could be made out in the sac.

The feet were in the position of ordinary typical equinovarus of rather an extreme degree. The test of power in the lower extremities was rather unsatisfactory. There did not appear to be any incontinence of feces, but the condition of the bladder could not be determined with certainty. There was a small umbilical hernia.

The temperature was taken for the first two days, but, as it remained normal, the record of the temperature was

then discontinued. The tumor was covered with sub-iodide of bismuth; over this was laid a compress covered with vaseline, and over this a large pad of absorbent cotton, held in place with a flannel roller applied so as to make gentle uniform pressure over the sac.

During the first two weeks the child was quiet and slept most of the time, and presented nothing abnormal in the way of symptoms. At the end of this time there was no longer any doubt regarding paraplegia. It was nearly complete; the thighs were habitually slightly flexed, while the legs were extended. Sensation was still doubtful. There was no incontinence of fæces, and retention or incontinence of urine was apparently absent.

At this date the tumor was noted to be perceptibly smaller, its surface somewhat wrinkled, and its translucency was very imperfect. The wall of the sac seemed to be thickened. On account of these changes in the sac, it was decided to postpone any operative procedures.

During the third week the general nutrition failed quite rapidly. The infant screamed much of the time and was quite restless. The temperature record was not kept, but it did not seem to be above the normal. There was no opisthotonos or vomiting; the bowels were regular and food was taken regularly.

Early in the morning of March 21st the infant was found cyanotic, with a very feeble pulse, and it died quietly a few hours later. There were essentially no symptoms during the last week, except those of rather marked irritability and failing nutrition.

AUTOPSY (three and a half hours after death).—There was nothing abnormal about the thoracic or abdominal viscera. Head of normal size; the fontanelles not bulging; slight overlapping of the cranial bones.

Brain.—Five ounces of rather thin yellow pus escaped from the lateral ventricles while the brain was being taken out. The whole cerebrum was rather soft; the convolutions were not markedly flattened. The pia mater of the convexity seemed normal. Its vessels were filled, but not distended, and there were no evidences of meningitis. The

sinuses were free of thrombi. At the base of the brain there was quite a thick ($\frac{1}{2}$ mm.) yellowish pellicle in the median line of the anterior surface of the medulla, which spread out in a thin layer, on either side, over the adjacent surface of the cerebellum, about one-half of which was thus coated. There was a similar pellicle on the optic tracts, about the optic commissure, and over a small area of the median anterior surface of the pons.

The Ventricles.—The lateral ventricles were very greatly dilated, measuring together $8 \times 6 \times 3\frac{1}{2}$ cm. in diameter, while in the normal brain of a child of the same age they measure together about $\frac{1}{2} \times 5 \times 2$ cm. The volume of the lateral ventricles in this case must have been at least forty or fifty times their volume in a normal brain of the same age.

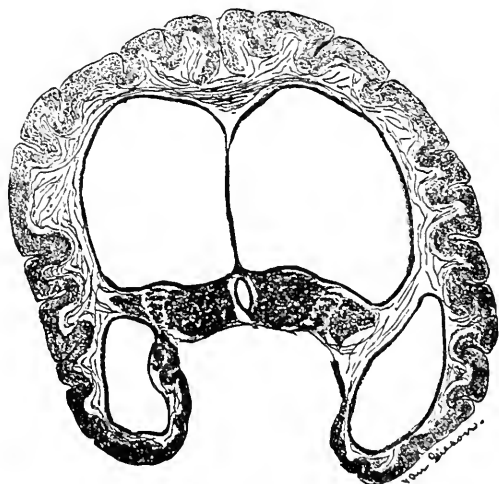


FIG. 1.—Transverse vertical section through the brain at the junction of the anterior $\frac{3}{5}$ and posterior $\frac{2}{5}$. The shaded zone lining the ventricles indicates the zone of granulation tissue—about $\frac{2}{3}$ of natural size?

In the section of the brain, shown in Fig. 1, the posterior horns measured 3 cm. in diameter. While the ventricles are thus enormously dilated, there is no corresponding expansion in diameters of the brain; the outer surface of the brain is not increased: the brain itself was normal in size, or perhaps a trifle smaller than usual. The increase of the volume of

the ventricles is formed apparently at the expense of the subjacent surrounding brain-tissue or by a condensation of the latter. The convolutions with their white matter are in places reduced to a mere shell in thickness (at the posterior and descending horns), measuring but 2 to 3 mm., about one-tenth of the thickness of the corresponding portion of the normal brain at the same age. The average thickness of the cortex is about 8 mm. The basal ganglia are only about half their normal vertical diameter and are soft. The lateral ventricles and their horns and the third ventricle are everywhere lined with a thin (1 mm. in diameter) translucent red membrane, having the appearance of granulation-tissue, which can be readily stripped up from the subjacent brain-tissue. Lying over this reddish membrane in many places are extensive flocculi of yellowish white material. The fourth ventricle is normal, except that its floor is congested. The medulla is much congested.

The meninges and substance of the *spinal cord* are uniformly congested. Lying on the surface of the pia mater are scattered yellowish white pellicles, which are most extensive at the lower part of the cord.

The *sac* contained about half an ounce of pus, and its walls were thickened by a thick (2 to 5 mm.) yellowish white layer lining its internal surface. The cauda equina entered the sac and passed along its walls in the usual way.

MICROSCOPICAL EXAMINATION (by Dr. van Gieson).—*The Wall of the Sac*: With the exception of a few scattered heaps of necrotic cells of the Rete Malpighii, the epidermis is absent from the oval central cicatrix of the sac (Fig. 2, *E—E*). In this central portion the papillary and more superficial portions of the derma are densely crowded with small round cells. The subcutaneous connective tissue is also infiltrated, but less extensively, with small round and elongated cells (Fig. 2, *b—c*). Both of these layers are much congested, the subcutaneous connective tissue containing the larger and more irregular vessels.

Beneath or internal to the subcutaneous connective tissue of the skin of the sac is a zone (Fig. 2, *a*) of irregular thickness, with a ragged free internal border, composed of frag-

mentary nuclei, necrotic material, and a scanty amount of fibrin lying on or near the free margin. In this zone the strata near the free margin are more distinctly necrotic than the deepest laminæ bordering the subcutaneous connective tissue; these have the appearance of granulation-tissue in a somewhat necrotic condition. This zone contains great numbers of bacteria of three kinds: a few short, thick bacilli, some large cocci, and the most numerous predominating form—small cocci in chains and clusters. The bacteria are most numerous near the free margin of the semi-necrotic zone (Fig. 2, *a*). This is the zone which appeared at the autopsy as the yellowish shreddy membrane coating the inner surface of the sac.

At the autopsy no apertures or solutions of continuity were noticed in the skin of the sac, excepting the excoriation of its central portion; but in the sections from this region in two places, microscopic plug-like continuations of the necrotic zone extend through the entire thickness of the excoriated central portion of the skin of the sac and communicate with the outer surface of the skin, as shown in Fig. 2, *d*. These two minute plugs of necrotic tissue extending through the thickness of the skin contain many of the small cocci, which are also attached to the outer surface of the plugs, and to the outer surface of the skin bordering on the plugs (Fig. 2, *d*).

At the edges of the excoriated central portion of the sac, the epidermis is present and the inflammatory changes are less intense, but the necrotic membrane with its bacteria continues over the whole inner surface of the sac wall.

The Central Nervous System.—The inner surface of the *dura mater spinalis*, in places in the dorsal and lumbosacral regions, is coated with a thin layer of red blood-cells with a little fibrin. The *pia mater spinalis* has congested vessels, and is infiltrated to a moderate degree with small round cells. The *pia mater* passing into the anterior fissure is similarly very abundantly infiltrated (except in the upper cervical region) so that the cell exudation quite uniformly distends the bottom of the anterior fissure (Fig. 3, *A*), and fills up the two large canals in the anterior com-

missure on either side of the central canal (Fig. 3, *c. c.*). These two canals in the anterior commissure, through which the sulco-commissural branches of the anterior spinal vessels pass obliquely into the gray matter, lies so close to the dilated central canal, as to furnish a passage-way for the exudation in the anterior fissure into the central canal (Fig. 3).

In several places in the lumbo-sacral region the exudation in the anterior fissure and in the pervascular spaces communicating with the fissure was so abundant as to have broken through the anterior commissure into the enlarged central canal.

As in the anterior fissure, the congested vessels of the posterior septum, in places, are surrounded by investments of small round cells, which are continuous with similar cells in the central canal (Fig. 3). There are scattered pellicles of partially necrotic material upon the surface of the pia mater which are most extensive and form an almost continuous layer upon the posterior pial surface except in the upper cervical region.

The vessels of the *spinal cord* are greatly congested, especially in the gray matter. The *central canal* is large, measuring, in general, about 1 mm. in diameter. It is filled up with leucocytes, necrotic or granular material, and some fluid and fat globules (Fig. 3). A good deal of the epithelium of the central canal has been desquamated or destroyed. There is hardly any evidence of inflammation of the substance of the spinal cord, excepting some perivascular heaps of leucocytes just about the margin of the central canal, and these changes in the wall of the central canal and its contents seem to be due for the most part to the extension of the spinal meningitis exudation through the anterior commissure, and along the paths of the vessels of the anterior fissure and posterior septum.

In the upper cervical region the central canal is normal, the congestion of the gray matter is less intense, and the spinal meningitis less in degree, and in places almost entirely absent. Just before entering the fourth ventricle

the central canal is again dilated and filled up with necrotic and exudative products.

In sections stained with Gram's method, and examined with the Zeiss $\frac{1}{12}$ oil immersion lens, small cocci, like the predominant form in the sac wall, are present in the pia mater, in the anterior fissure and its communicating channels in the anterior commissure, in the exudation plugging the central canal, and in the pellicles lying over the pia mater (Fig. 3).

The *flake or pellicle of pus on the pons* described in the autopsy notes contains small cocci like those in the cord and sac wall.

The walls of the *third ventricle*, the *upper portion of the aqueduct of Sylvius* and of the *lateral ventricles* are everywhere uniformly changed. They are lined by a zone of granulation tissue, bordered internally by ragged necrotic fibrinous layer of variable thickness containing very great numbers of the small cocci similar to those in the cord and sac wall (Fig. 6, *A and B*).

This fibrinous necrotic zone corresponds to the yellowish pellicle, and the granulation tissue zone to the reddish membrane subjacent to the pellicle mentioned above in the gross description of the ventricles. The vessels of the choroid plexus of the lateral ventricles are congested and their meshes filled with small round cells. The *fourth ventricle* is normal, except that the choroid plexus vessels are congested, and there is some proliferation of the choroidal epithelium.

The *pia mater of the convexity* shows in most places a moderate degree of exudative meningitis.

Anatomical Diagnosis.—Spina bifida with hydromyelia. Suppurative inflammation of the wall of the sac. Purulent spinal meningitis, with the exudation extending into the central canal. Suppurative ependymitis of the lateral and third ventricles, exudative cerebral meningitis.

Remarks.—The bacteria were, unfortunately, not identified by culture, but the small cocci are presumably some form of pyogenic bacteria, which entered the exposed surface of the skin of the sac, producing an inflammation of

the wall, and extending along the central nervous system, excited the lesions in the spinal cord and brain.

The *absence* of the *physical signs* of congenital *hydrocephalus* is remarkable. Yet the greater part of the fluid accumulation within the ventricles appears to have been formed at birth, and does not seem to have been caused by the suppurative endymitis. For in this case the large intra-ventricular collection would have been formed so rapidly—within three weeks—that apparently it would have necessarily produced enlargement of the head and distention of the sutures or fontanelles. Probably at birth the ventricles were nearly as large as at the autopsy, and were filled with clear fluid; then after the lodgement of the cocci in the walls of the ventricles, the exudative purulent endymitis occurred, which increased the intra-ventricular contents somewhat and converted the clear fluid of the ventricles into a sero-purulent fluid.

The morphological identity of the small cocci in the different regions and the absence in the brain and cord of the other two forms of bacteria in the sac, indicate the small cocci as the real pathogenic agents and that the associated bacteria are merely contaminating forms. As far as can be determined from their dimensions and grouping, and by comparing them with other known species of pyogenic micro-cocci in control sections, the small cocci in this case are thought to be the *streptococci pyogenes*.

The plugs of necrotic tissue extending through the skin of the sac are rather suggestive as to the probable points of entrance of the cocci.

A very interesting feature of the case bearing on the pathology of the hydro-myelia and syringo-myelia is the demonstration of how readily an exudation of the pia mater spinalis in the anterior fissure may communicate with an imperfectly closed central canal, by following the perivascular spaces of the vessels entering the cord, or by breaking through the anterior commissure.

*Laboratory of the College of Physicians and Surgeons,
437 West 59th Street, New York, May 18, 1890.*

A STUDY OF HEAT-PRODUCTION AND HEAT-DISSIPATION IN THE NORMAL AND FEBRILE STATES.¹

By WILLIAM A. CARTER, M.D.

(From Private Laboratory of Dr. Isaac Ott.)

IT is a well-known fact that there is a diurnal rhythm in the growth of plants. They grow at night and remain inactive during the day.

M. Fredericq, on studying the absorption of oxygen by a normal man from 8 A. M. to 7 P. M. found two maxima—at 10 A. M. and 3 P. M. Upon a starved man these maxima did not exist, but the curve fell regularly.

Prof. Jürgensen has shown the existence of a diurnal rhythm in the temperature of man. The temperature reaches its maximum at 9 P. M. and its minimum at 7 A. M. He divides the 24 hours into two periods—the diurnal from 7 A. M. to 9 P. M. (the mean temperature being 99.2° F.) and the nocturnal from 9 P. M. to 7 A. M. (the mean temperature being 98.49° F.). He found too that this rhythm is present in fever, and is not affected by cold baths, starvation or muscular exercise.

Prof. H. C. Wood, in his work on "Fever," says: "There probably exists a rhythm of heat-production and heat-dissipation, the maximum and minimum of which occur synchronously with the maximum and minimum of animal temperature."

¹ This thesis was awarded the Medical News Prize of University of Penna., 1890.

P. Langlois² made calorimetrical experiments on infants from 8 A. M. to 5 P. M., and found the heat-production attained its maximum at 3 P. M.

This is all the work that has been done on this subject to the best of my knowledge. This paper gives the results of experiments on rabbits, cats and dogs, showing the heat-production and heat-dissipation during the 24 hours. At no time was the animal kept in the calorimeter longer than an hour. Several of the experiments were performed with the improved calorimeter of this laboratory, whose error is only 5.4%. The observations were all made either four or six hours apart, so that the calorimeter could not bring the heat-dissipation into an abnormal condition. That there is much more heat produced in an animal after a meal than before, although the temperature remains the same, is a well-known fact. The heat-production of an animal is probably increased 35%-40% by the ingestion of food.³ In order to eliminate any such influences, the animal was starved three days before the experiment began. Prof. Chittenden, of Yale, starves his animals three days in order that all food-stuffs taken into the organism might have time to be assimilated and eliminated. Three days seem ample time for the disappearance of food, so that in these experiments all the metabolic changes by which heat was produced took place in the stored-up material of the organism. Strong, healthy rabbits were used, and when possible, white ones, for they seem to be more susceptible to any changes in heat-regulation than do black ones. This fact has been pointed out by Prof. Wm. H. Welch.⁴ The first three experiments lasted 48 hours. The heat-production decreased steadily toward the end of the experiment, caused, no doubt, by the long starvation. In order to avoid this, the other experiments only lasted 24 hours—the observations being taken every four hours.

Out of 20 days the maximum heat-production came at 7 A. M. three times, at 11 A. M. twice, at 3 P. M. five times,

² *Journal de L'Anatomie et de la Physiologie*, Paris, 1887.

³ Langlois, *Journ. de L'Anat. et de la Physiol.*, Paris, 1887.

⁴ Cartwright Lectures on The General Pathology of Fever.

at 7 P. M. three times, at 11 P. M. four times, and at 3 A. M. three times, or ten times during the day and ten times at night.

The minimum occurs at 7 A. M. seven times, at 11 A. M. four times, at 3 P. M. once, at 7 P. M. three times, at 11 P. M. twice, and at 3 A. M. three times, or twelve times during the day and eight times at night.

The minimum came at 7 A. M. most often, but in five of these cases the experiments ended at that hour, and as the animal had been starved four days, it is obvious why the minimum should occur at that hour.

In the experiments in which the same animal was used twice, the maximum and minimum did not appear twice at the same time.

These results seem to prove the non-existence of any diurnal rhythm of heat-production and heat-dissipation.

The heat-production and heat-dissipation are not affected enough by external circumstances to produce any diurnal rhythm, but being under the control of a nervous mechanism, and having for their one great object the maintenance of temperature, they run a very unsteady course, being high at one time and low at another. Of all external circumstances which influence heat-production and heat-dissipation, external temperature seems to have more effect than any other. Prof. Richet⁵ made 110 experiments to find the relation of external temperature to heat-production. He found a maximum heat-production when the external temperature was about 55.4° – 57.2° F., and any temperature above or below this point decreased the heat-production.

We find the maximum temperature at 7 A. M. once, at 11 A. M. once, at 3 P. M. once, at 7 P. M. fourteen times, and at 11 P. M. three times, or three times during the day and seventeen times at night.

The minimum came at 7 A. M. eight times, at 11 A. M. seventeen times, at 3 P. M. twice, at 7 P. M. once, and at 11 P. M. twice, or seventeen times during the day and three times at night.

These results show a distinct rhythm of temperature, the

⁵ Acad. des Sciences, June, 1885.

maximum occurring in the evening (7 P. M.—11 P. M.) and the minimum in the morning (7 A. M.—11 A. M.). This rhythm in the rabbit, cat and dog is the same as Prof. Jürgensen found in man.

The maximum and minimum of heat-production and heat-dissipation do not occur synchronously with the maximum and minimum of animal temperature. The two are entirely independent of each other.

Krieger has shown when the individual sleeps during the day and works during the night the typical course of the temperature is reversed. This statement has been confirmed by observations made at the suggestion of Prof. Huguenin, of Zürich, by Herr Buchser, an engineer of Rodi, who is accustomed to sleep during the day and work at night. As his tables show, his morning temperature oscillates between 37° and 37.5° C., while the evening record is between 36.6° and 37° C., averaging 36.8° . Maurel ascribes this vesperal rhythm to three causes—food, light and movement—and of these food is the most important. This factor is totally disproved in my experiments, as the animals had fasted three days, yet the vesperal exacerbation ensued. The worker at night reverses this rhythm, and here light could not be the factor, unless it is the artificial light, which would be hardly sufficient. From these results the conclusion ensues that it must be due to an action of the nervous system and probably to the activity of the thermotaxic centres.

This same absence of rhythm of heat-production was found in three experiments (lasting 48 hours) on animals that were constantly supplied with food. Here, as is to be expected, the variations were greater than in the starved animal. The same rhythm of temperature as exists in the starved animal was present.

To study the rhythm in fever it was necessary to produce a more steady fever than the septic fever. Each animal was starved two days. Then the centre of Eulenberg and Landois (at the junction of the cruciate and great longitudinal fissure) was destroyed and the animal starved another day. By this time the fever was fully established,

and the experiment upon heat-production was begun.

The maximum came at 11 A. M. five times, at 3 P. M. three times, at 7 P. M. once, at 3 A. M. three times, or eight times during the day and four times at night.

The minimum came at 11 A. M. twice, at 3 P. M. twice, at 7 P. M. once, at 11 P. M. twice, at 3 A. M. twice, and at 7 A. M. three times, or seven times during the day and five times at night.

There appears here a slight tendency for the maximum to recur about mid-day or shortly after, but in some of these cases the experiments began at that hour, and besides, the experiments are entirely too few to warrant any statement that there is a diurnal rhythm.

The maximum temperature occurs at 11 A. M. twice, at 3 P. M. twice, at 7 P. M. five times, and at 7 A. M. three times.

The minimum comes at 11 A. M. three times, at 3 P. M. twice, at 11 P. M. three times, at 3 A. M. once, and at 7 A. M. three times.

These results show a much greater variation in the temperature of the fevered animal than in the normal animal, although it is probable that the same rhythm exists.

The heat-production and temperature are entirely independent of each other.

Below is given a table showing the average weight of each animal, the average heat-production and heat-dissipation and average rectal temperature of each experiment.

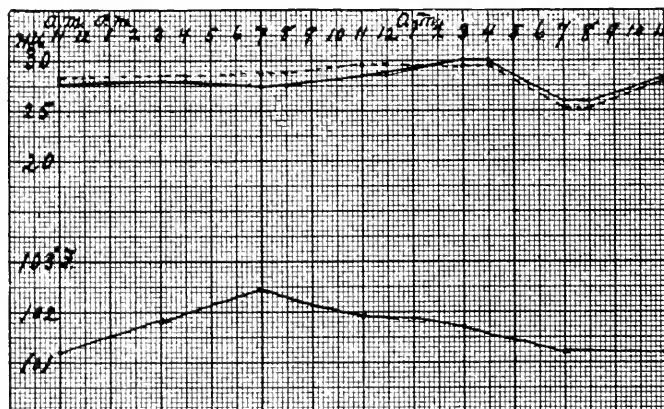
It will be seen that generally the greater the weight the greater is the heat production.⁶ The animal temperature does not seem to have much effect on the heat-production, and the changes of the two are entirely independent of each other in all the experiments. The external temperature, however, undoubtedly affects the heat-production.

⁶ Since this was written Prof. E. T. Reichert has published a paper showing the same result—University Medical Magazine, February, 1890.

Ex.			Average Wt., Average		Average	Average
			lbs.	H. P.	H. D.	H. T.
1.—	Starved Rabbit,		4.17	30.02	28.41	102.16
2.—	"	"	5.47	36.70	32.19	103.16
3.—	"	"	5.33	35.27	38.05	101.15
4.—	"	"	3.70	19.86	19.81	102.16
6.—	"	Cat,	3.02	22.63	23.23	101.33
7.—	"	"	4.91	24.91	26.21	101.00
8.—	"	Dog,	10.71	51.08	50.78	100.50
9.—	"	Rabbit,	3.33	28.06	28.92	102.13
10.—	"	"	2.77	17.47	18.48	101.46
11.—	"	Dog,	10.93	47.54	45.30	100.85
12.—	"	Rabbit,	2.68	21.57	22.59	103.03
13.—	"	Dog,	10.65	49.87	49.35	101.05
14.—	"	Rabbit,	3.27	22.22	23.08	101.95
15.—	"	"	3.44	21.64	22.31	102.60
16.—	"	"	3.42	17.42	18.07	102.05
17.—	"	"	3.10	24.73	24.62	103.30
18.—	"	"	3.62	23.03	24.06	101.33
19.—	Fevered Rabbit,		4.25	27.01	24.19	103.22
20.—	"	"	5.02	36.71	33.10	103.68
21.—	"	"	5.47	35.09	29.46	103.10
22.—	"	Cat,	6.42	35.82	38.03	103.95
23.—	"	"	6.21	36.67	38.82	103.25
24.—	"	"	3.41	22.67	24.33	103.25
25.—	"	"	4.49	32.96	35.47	103.40
26.—	"	"	4.42	21.96	22.10	102.14
27.—	"	"	2.46	21.81	22.14	104.15
28.—	"	"	2.38	21.91	22.23	105.06
29.—	"	"	6.09	34.35	37.89	104.21
30.—	"	"	5.80	28.14	32.32	105.66
31.—	"	"	5.49	28.98	32.24	105.91
32.—	"	"	3.95	24.08	25.46	105.46
33.—	"	"	5.55	37.62	40.55	104.58

Below are two composite curves showing the average heat-production and heat-dissipation and rectal temperature. The continuous line represents heat-production, the dotted line heat-dissipation, and the line below the rectal temperature at the *beginning* of each observation, when it is not affected by being in the calorimeter. The temperature

at the end of the hour is not given, so that what at first seems impossible is only apparent on comparing the changes of relation of heat-production and heat-dissipation with the changes of temperature.



Composite Curve showing the average of twenty experiments on starved animals.



Composite Curve showing the average of twelve experiments on febrile animals.

TREATMENT OF EPILEPSY.¹

BY PHILIP ZENNER, A.M., M.D., CINCINNATI, O.,

Clinical Lecturer on Diseases of the Nervous System in the Medical College of Ohio.

EPILEPSY is no longer the *morbis sacer*, the scourge of the gods of the ancients, against which therapeutic measures are necessarily futile, but nevertheless its oftentimes intractableness to treatment creates the too common impression that treatment is of little avail.

In truth, there are few chronic diseases in which therapeutic measures have equal power. But in order that their full power should be obtained, the treatment must be carefully adjusted and faithfully carried out; and this requires great care and patience on the part of the physician, and full confidence as well as untiring perseverance on the part of the patient. For this very reason pessimistic views on the part of the physician are exceedingly unfortunate, for they lead to lax methods of treatment, and inspire little confidence in the patient, so that failure is almost a certainty.

Scepticism, here, is but the natural reaction from the other extreme. For, as in many other intractable diseases, innumerable remedies have been vaunted for their high curative powers, which, on further trial, were found to be altogether, or almost, valueless.

Some of the discrepancies of experience are really surprising. For instance, that great physician Schroeder v. d. Kolk, believed counter-irritation to be an almost infallible remedy, and to-day it is scarcely used, while Herpin reported a cure of 28 out of 42 cases with oxide of zinc, a success unapproached, otherwise, in the treatment of this malady, and yet oxide of zinc is rarely prescribed for epilepsy now. These successful results may have been largely due to a fortunate selection of cases; at least in the case of

¹A paper read before the Mississippi Valley Medical Association, at Louisville, Ky., Oct. 10, 1890.

Herpin, the results were partly confirmed by Voison¹ who investigated the cases reported by H. long after his death, and found many of them still cured of their epilepsy. But such favorable results, must, for the larger part, be attributed to hasty and inadequate observations, colored, furthermore, by ardent enthusiasm.

In order to duly appreciate the value of drugs, we must learn what we can of the natural course of the disease, and how it is effected by other influences.

As in other diseases, there is a natural tendency to recovery. According to Nothnagel, nature, unassisted by art, cures from 4 to 5 per cent. of all cases. Disease, especially acute disease, often cures epilepsy.

A temporary favorable turn in the disease is far more common than a definite cure. It is not uncommon for an injury or a wound or a surgical operation, especially a capital operation, to be followed by a reduction in the number of fits, or their entire cessation for a longer or shorter period of time. This fact should lead to caution in pronouncing a cure shortly after an operation for traumatic epilepsy—a rule rarely heeded.

Acute disease often causes a temporary suspension of the fits—a fortunate result, as it may, at the same time, necessitate the suspension of the anti-epileptic remedies. A change of scene or place, for instance entering a hospital or asylum, often causes a temporary improvement, as does frequently the administration of a new remedy, whatever it may be.

Such facts should make us cautious in drawing hasty conclusions as to the effect of drugs or modes of treatment at an early period. The permanent effects alone can be a guide to their efficacy.

The study of etiology is exceedingly important in approaching the subject of treatment. Two distinct and different conditions are to be specially considered; firstly, an hereditary or acquired constitutional vice, which predisposes to the development of the disease, or perhaps it

¹ Bull., gener. de therap. lxxviii., 193.

should be considered the disease itself ; and secondly, local diseased processes, which, reflexly, act as exciting causes of attacks, and, in some instances, have caused the underlying constitutional vice. The first of these conditions we must suppose to be always present, and therefore the more important of the two, but the second has such practical bearings that it is of much consequence in treatment.

The local processes which act as peripheral, reflex sources of irritation, are of the most diverse character. The most common are injuries or wounds, especially injuries about the head, or wounds of nerves. Of local diseases we may mention : first, disorders of the alimentary tract. In infants these are very common, perhaps the most common cause of convulsive seizures. In this respect they play a less important rôle in adult life, though they may do so in cases where least expected. I will briefly mention an interesting case bearing on this point.

A man, now 50 years of age, of a healthy family, usually quite healthy and of a phlegmatic temperament, had in the course of 18 months ten convulsive seizures, which occurred always in sleep, and became more severe and frequent toward the end of this period. They ceased on the discovery and expulsion of a tapeworm. Six months ago, after a disappearance of five years, a convulsion again appeared and was followed by three or four others, in as many months. The man, on his own responsibility, and guided only by his former experience, took a taenicide, which was followed by the expulsion of a tapeworm.

The next source of irritation to be mentioned is in the genito-urinary system. In the male, phymosis, in the female, ovarian disease, are the ailments which here played the most prominent parts. Of late years much attention has been called to eye-strain, from lack of balance of the extrinsic muscles, or anomalies of refraction, as a reflex source of nervous disease. Though its influence has, doubtless, been overestimated on some sides, it is a condition which should be carefully sought for, and remedied when

found. In addition to the above I will only mention disease of the nasal mucous membrane, the removal of which has, in some instances, been followed by disappearance of the epilepsy.

All these peripheral sources of irritation—and I have only given the most prominent ones—deserve careful consideration. But they may lead into error in several ways. There may be a tendency to give them a much more widespread importance than is their due, and occasionally harm is done by the local treatment. This is especially true of gynæcological treatment, so often resorted to merely on account of the presence of nervous disease, to the very frequent detriment of the patient. This overestimation of the importance of reflex sources of disease is, on the other hand, liable to lead others to the opposite extreme, so that no attention is paid them at all—an equally fatal error. Each case should be carefully examined and treated on its own merits, quite regardless of what may have been found in other cases. But it must especially be emphasized that the removal of local disease must always be considered but a part of the treatment in cases of epilepsy. It must always be remembered that in addition to the local disease there is the constitutional vice; or, if the former was the original cause of the epilepsy, the latter may now have gained an independent and stable existence.

In every case, therefore, whether a reflex source of irritation was found and remedied or not, careful attention must be given to the constitutional condition. This comprises all measures which tend to improve the general condition and tone up the nervous system. Among these we may briefly mention, avoidance of excitement, the leading of a quiet, peaceful life, outdoor life when possible, an abundance of sleep and light, nourishing food. Of special therapeutic measures I will only mention the perhaps most important, hydrotherapy. Of this form of treatment, properly regulated cold baths and sponge baths are most applicable.

In all cases of epilepsy the treatment thus far outlined should be instituted; that is, the removal of peripheral

sources of irritation when found. and the general treatment to improve the tone of the nervous system. In some instances this treatment will be sufficient. Often patients come to us with the history of one, or only a few convulsions, in which the age and other conditions lead to the diagnosis of beginning idiopathic epilepsy. In cases of that kind, I have been accustomed to institute the treatment above mentioned, and, in quite a number, no convulsion has since appeared. But in cases of chronic epilepsy, we must resort, in addition, to anti-epileptic drugs. The number of such drugs which have been used is very great. Of those which have come down to us from the time before the bromides came into use, the most prominent are oxide of zinc, nitrate of silver, digitalis, and belladonna. Digitalis is, probably, of value in cases of weak heart and feeble circulation, and is still not infrequently used in such cases. Belladonna is also much in use at the present time, and is frequently a valuable adjunct to the bromide treatment. Trousseau, who wrote before the bromides came into use, considered belladonna the most valuable medicine in the treatment of epilepsy. He speaks of it as effecting a cure in rare instances, and as frequently exerting a powerful control over the disease. His method of administering the drug was to begin with 1-100 gr. of atropia once a day, and each month to increase the daily dose by 1-100 gr. until from five to twenty times that quantity was taken daily. In some instances he combined the belladonna with nitrate of silver, sulphate of copper, and oxide of zinc, successively.

With the introduction of the bromides came a new era in the treatment of epilepsy. It enabled physicians to obtain a control of the disease hitherto unapproachable. That it has not gained a much greater success than has already been accorded it is owing to lax methods on the part of physicians, and lack of perseverance on the part of patients. It is especially necessary in this mode of treatment to study carefully the patient, and the effect of the drug upon him. Occasionally a case is found in which the treatment is not applicable, but in the great majority it can be used with a greater or less degree of efficacy. As regards the dose, no

definite size can be mentioned. Each case is to be individualized. Some will bear the smallest dose badly, while in others four or five drachms can be taken daily for an indefinite period. It is well to begin with about fifteen grains two or three times a day, and very slowly increase the dose until the desired effect is produced. But two things should limit the size of the dose—the complete control of the disease, or the production of bromism. After reaching the maximum dose which can be well borne by the patient, or that which holds the disease in complete check, it should be continued in the same doses for a long period of time. If the disease is held only partly in check, it may be necessary to continue the dose with small modifications for years or for life. If the convulsions have altogether ceased, the medicine should still be continued for several years and then the dose gradually reduced until it be suspended altogether.² Sometimes, after eight or nine months, it may be safe to lessen the dose, but this must be done with great caution, and, if ill effects occur, the maximum dose must immediately be resumed. Sometimes the system seems to become accustomed to the drug, and the latter loses its control. If so, the dose must be increased.

Usually, the medicine should be given two or three times a day in equal quantities from day to day, but when the paroxysm occurs at stated periods, there may be some modifications in the mode of administration. If the convulsion usually occurs at a certain hour of the day, the whole daily quantity of medicine should be given a few hours before the expected time. If it occurs especially at stated times, for example, at the menstrual period, the drug

² The custom of Legrand du Saulle (Dujardin Beaumetz, *Leçons de Clinique Thérapeutique*, iii., 193) is, after one year of freedom from convulsions, to give the bromide only six days out of seven; after fifteen months, five days out of seven; at the end of eighteen months, only four days out of seven; and after two years, only three days out of seven.

Seguin (*N. Y. Med. Jour.*, Apr. 5, 1890), begins a gradual reduction when three years have passed without any manifestation of the disease, giving gradually smaller doses until the end of the fourth year, when the medicine may be discontinued altogether.

should be given in large doses a few days preceding the expected attack, and in much smaller doses in the free interval.

As the bromide treatment must be continued for years, the ill effects of the drug must be carefully studied, that they may be avoided as far as possible. These are : 1. Disturbances of the digestive tract. These can usually be avoided by giving the medicine largely diluted in water, preferably alkaline water, at least one-half gobletful, and if the dose be large, a gobletful. 2. Cutaneous eruptions, especially acne. In some persons the susceptibility to serious cutaneous eruptions is so great as to thwart this method of treatment altogether, but usually it need not interfere with the treatment. To some extent this trouble can be kept under control by arsenic. 3. A blunted condition of the will and intellect is usually more or less marked. If this increases to a great degree, it may be necessary to temporarily diminish the dose, though there often appears to be an adjustment, and the symptoms subside somewhat without reduction of the dose. Often, after the fits have been brought under control, the patient's disposition changes ; there is great irritability, etc. But in this case, too, an adjustment seems to take place between the system and the medicine, and the unpleasant manifestations disappear without changing the dose. 4. The bromides sometimes cause impairment of nutrition, so that in badly nourished individuals it must be used cautiously. The use of cod liver oil, iron, or other tonics, and especially an abundance of nutritious food, may help us over this difficulty. The same tonic course is usually indicated in cases of epilepsy. A weak heart, or valvular disease, also demands caution in the use of the bromides. Digitalis is, in such cases, a valuable adjuvant.

Legrand du Saulle termed the bromides a muzzle for epilepsy. The term is well applied. The drug reduces the excitability of the nervous system, and thereby holds the disease in check. In the progress of the treatment we must be very careful in lessening its hold. A sudden withdrawal of the drug is very likely to be followed by a return

of the fits, sometimes in large number. Acute diseases sometimes necessitate a reduction or entire suspension of the drug, but, fortunately, convulsions are less likely to occur at such times. When the condition of the patient or the effect of the drug makes it necessary to reduce the dose, the decrease should be substituted by some other drug having anti-convulsive properties. Some of these I have already mentioned, the most valuable of which is belladonna. Chloral has been highly recommended by Seguin³ as a partial substitute for the bromides, and he believes it to have equal anti-convulsive powers. He substitutes from one-fifth to one-half of the bromide dose, with an equal quantity of chloral, whenever necessary to diminish the former on account of severe cutaneous eruptions or the like. He thinks that the chloral affects the cardiac nerves and cerebral cortex less unfavorably than the bromide, and that the two drugs together have a more powerful control over epilepsy than the bromide alone. He states that since using this combination he has not seen the mental disorder arise which in rare instances follows the entire suppression of convulsions.

The success of the bromide treatment is dependent on the constant watchfulness of the physician, and untiring perseverance of the patient. There are few patients who cannot bear the medicine, if care be taken in its administration; few to whom it will not be of some benefit; while in a large per cent. of cases it will prolong the interval between the attacks to such an extent as to lead to a practical cure, and in rare instances an actual cure is effected. Failures are commonly due to lax methods on the part of physicians, to inattention to details, or to lack of perseverance on the part of patients, who require every encouragement to carry them over the many difficulties and disappointments obstructing the way to improvement or cure.

I will add a word as to the different kinds of bromides. Bromide of potassium is the form most commonly in use, and has a strong controlling power over the disease, but

³ N. Y. Med. Jour., Apr. 5, 1890.

bromide of sodium is selected in preference by many physicians, because it produces less irritating effects on the digestive tract, and less depressing effects on the heart. A common mode is to administer a mixture of the three bromides of potassium, sodium and ammonium. It has been supposed by some that the efficacy of the preparation was according to the proportion of bromine present, and as the soda salt has 77.7 per cent., and the potash salt only 67.2 per cent.; the former should, therefore, have greater efficacy than the latter. But this can not be held as a safe rule. For instance, the bromide of gold, whose efficacy in epilepsy has recently been lauded by a number of St. Petersburg physicians,⁴ contains only 55 per cent. bromine, and is given in but 1-6 to 1-5 grain doses. A few years ago bromide of nickel was highly recommended by DeCosta,⁵ as having very favorable effects in 5 to 10 grain doses, and being very well borne by the stomach. In the hands of some other observers⁶ the drug did not have equal success.

I will speak very briefly of a few other drugs, of which more or less favorable results have been reported very recently.

Borax has been brought forward in medical journals by quite a number of observers in the past year, though it is not new in the treatment of epilepsy. The most extensive article on the subject is by Taylor and Russell,⁷ in the *Lancet* of May, 1890. They obtained favorable effects in twenty cases, in doses varying from 10 grains to a drachm, though, they think, a 30-grain dose need not be exceeded. They found the continued use of the drug liable to produce ill effects, which may necessitate a suspension of treatment. These are chiefly, cutaneous eruptions, cracking of lips and peeling of the tongue, disturbances in the alimentary tract, and impaired general nutrition. These difficulties may be in part controlled, if not averted, by administration of arsenic and cod liver oil, and large dilution of the drug.

⁴ Med. Record, 1890, p. 183.

⁵ Med. News, 1883, p. 337.

⁶ Bourneville, *Le Progres Medical*, 1889, p. 497.

⁷ The *Lancet*, May, 1890.

Finley,⁸ Folsom⁹, Stewart,¹⁰ Diller, Bury¹¹ and Hill¹² have also reported favorable results, while Jones,¹³ who tried it in ten cases, and Pomeroy,¹⁴ in twenty, report complete failures. But both the latter applied the remedy to inmates of insane asylums—very unfavorable subjects for treatment. My own experience with this drug is not large. A number of years ago I tried it in some dispensary patients, but they soon disappeared from view. Within a few months I have prescribed it in quite a number of patients, with apparently favorable effects, but the time is too short to give any reliable information. The same is true of the reports above referred to. They can only be deemed incitives to further and longer continued trial of the drug.

Wildermuth¹⁵ has recently made extensive observations with amylene hydrate. His results were not favorable to its continued use in the treatment of epilepsy, but he found it to be unusually valuable in the status epilepticus.

Antipyrine and antifebrine have also been much employed in such cases, and with quite varying results. In my own hands, I have found them of some value in lessening the number of fits.

Among other drugs I will only mention simulo, osmic acid, and nitro-glycerine, which have been tried without much encouragement for their future use.

We may learn to look upon any of these drugs as reliable remedies, if, as is true of the bromides, they are able to hold the epileptic seizures in check indefinitely. It is even possible that some of them have a specific curative power, in special cases, in a way we do not understand.¹⁶

⁸ *Neurolog. Cent. Blt.*, 1889, p. 27.

⁹ *Ibid.*, 1887, p. 116.

¹⁰ *Brit. Med. Jour.*, April, 1890.

¹¹ *Canada Lancet*, August, 1890.

¹² *Med. News*, 1889, p. 512.

¹³ *Boston Med. and Surg. Jour.*, 1890, p. 36.

¹⁴ *Ibid.*, p. 119.

¹⁵ *Neurolog. Cent. Blt.*, 1889, p. 451.

¹⁶ Thomson's suggestion (*Journ. of Nerv. and Ment. Disease*, 1890, p. 246) that epileptic attacks are often due to absorption of ptomaines in the digestive tract is worthy of consideration in this connection.

But, at least, it is probable that some of these remedies may be of value in assisting the bromide treatment, either in the way of substituting the bromides temporarily, or being used in conjunction with them. Amadel¹⁷ seems to have proven the value of the combination of remedies. He combined the bromide with antipyrine, and no convulsion was observed in four months in the asylum at Cremona.

One word as to prognosis. The disease is usually the more amenable to treatment the shorter its duration and the fewer the number of attacks. Cases with only nocturnal attacks are usually more favorable than those with diurnal attacks, and those of *grand mal* more favorable than *petit mal*. Cases of *petit mal*, and especially, those with mental symptoms, are, usually, very intractable to treatment.

I must again emphasize, especially in connection with these cases, more difficult of management, the importance in each instance, of seeking, and where possible removing, reflex sources of irritation, and especially, applying the general treatment before mentioned.

We cannot complete a review of the present treatment of epilepsy without referring to its surgical treatment.

Some operative measures, such as laryngotomy, ligature of the carotids, indiscriminate castration, the amputation of a part where the aura began, fortunately belong to the past and need no further mention. Other surgical operations, which fulfil causal indications, such as the removal of cicatrices, of carious teeth, diseased bone, etc., also need no further mention.

Ligature of the vertebral arteries, one or both, first performed by Alexander, of Birmingham, 1880, deserves passing notice. So far as I know, the operation has been made by four operators in 39 cases. Of 35 cases collected, and mostly operated upon, by Alexander, he reports 8 cured, 11 improved, and 16 unchanged. Recently, Roman v Baracz¹⁸ reported four operations, and highly recommended the operation, though his own cases were reported too early to draw any conclusions from them. He thinks the results

¹⁷ Journ. Nerv. and Ment. Dis., 1890, p. 550.

¹⁸ Wiener Med. Wochenschrift, 1889, No. 7.

obtained may be due to including the sympathetic in the ligature, as the pupil is found to be contracted, and sometimes ptosis occurs on the side of the ligatured artery. Krüdener¹⁹ has concluded, from a number of experimental observations, that ligature of the vertebral or carotid arteries does not lessen the irritability of the cerebral cortex.

The surgical treatment of epilepsy comprises chiefly the operations about the head. Trephining for epilepsy is one of the oldest operations of surgery. At one time this was resorted to in any or all intractable cases. But these indiscriminate operations brought the procedure into discredit, so that for a long time it fell almost into disuse. It is again becoming more in vogue in recent years, and clearer ideas entertained as to its object, yet without the formulation of any distinct principles to determine its performance.

The success of this operation must be determined by experience, but the variance here is remarkable. Some of the best surgeons have been unable to report any cures. Thus von Bergman,²⁰ certainly an able and skilful operator, has obtained no complete success in any of his cases, while Walsham²¹ has carefully collected the reports of 82 cases, of which 47 were pronounced cured, and 13 improved. Others have collected tables in which the percentage of cures was much higher. The reasons for these great differences are that favorable cases are much more likely to be reported than unfavorable ones, and that the reports are usually made too early to permit of any definite conclusion. It has already been stated that any operation may cause a temporary disappearance of convulsions; so that immediate improvement can not be attributed to the curative power of the trephine. The convulsions must have been absent a long time before a cure is pronounced. Horsley gives five years as the limit, and even this lapse of time may not suffice for some cases, though a much shorter one might satisfy us in many instances.

¹⁹ *Neurolog. Cent. Blt.*, 1890, p. 174.

²⁰ *Surgical Treatment of Diseases of the Brain.*

²¹ *St. Bartholomew's Hosp. Reports*, vol. xix.

These strictures on ordinary reports are called for lest the latter should lead us to believe that operations accomplish very much more than they do, and should lead to their too indiscriminate resort.

While in very bad cases even a temporary benefit is not to be despised, and in the present incomplete state of knowledge an operation, without clear indications, in a desperate case can not be condemned, yet it is to the interest of science that such operations should only be made where clear indications are present. The facts that a capital operation is always a very grave matter to the patient and his family, that it is possible for such an operation to make the patient's condition worse, and that failures are likely to throw discredit on the operation, should lead us to the just-mentioned conclusion.

Then what are the indications for such operations?

The great majority of cases in which trephining is now resorted to are cases of traumatic epilepsy. Of this class, those cases must be considered appropriate for operation, in which there are circumscribed lesions, whose seat can be determined, and which are capable of removal. Thus defined, the operation has a very distinct and definite purpose—the removal of a source of irritation.

The lesions may or may not be found immediately underneath the seat of the trauma, and lesions in the cranial cavity may or may not be attended by apparent injury in the scalp or skull. Felizet's experiment with a skull filled with paraffine, in which a fall from a moderate height caused a flattening of the paraffine without fracture of the skull, shows how injury of the brain may be produced without apparent change in the bone. The experiment shows that the skull can be momentarily depressed and regain its former shape. The lesions which may be found in these cases are, besides cicatrices in the scalp and skull, adhesions between the membranes, gross meshed, cystoid scars, filling up flat, cup-shaped depressions on the surface of the brain; thin, rusty-colored, connective tissue layers, and thick sclerosed, and dense plaques in and on the cortex; splinters of the in-

ternal table, incorporated with the tissues underneath, and penetrating foreign bodies of all kinds.²²

To ascertain the seat of the lesions, two orders of symptoms are applicable ; firstly, the local symptoms or signs ; secondly, the nervous, or more properly, cortical symptoms. The local symptoms, apart from the cicatrix or depression in the bone, are local pain and tenderness, sometimes reflex symptoms, even convulsive movements or fully developed convulsions produced by pressure over the lesion. The mere recognition of the cicatrix or depression in the bone need not point to the intra-cranial source of irritation. A case of Denous²³ is very instructive. A fall on the head was followed by paralysis of right leg and left arm, a paresis still remaining, when, at the end of two years, left-sided convulsive seizures occurred. There was a cicatrix in the scalp, also a depression of bone on the left side of the skull, but D. paid no attention to these, trephining over the centre of the right fissure of Rolando. The remains of a linear fracture were found, and underneath this a meningo-encephalic collection. The operation resulted in a cure of the convulsive seizures and left-sided paralysis, the paralysis in the right side remaining as before.

The cortical symptoms may be monospasm (Jacksonian epilepsy) monoplegia, aphasia, irritative or parietic, sensory, ocular or aural symptoms, etc., according to the seat of lesion.

The object of the operation should be the removal of the source of irritation, the extirpation of the entire area of disease. In some cases the cicatrix in the scalp or skull may be the only offending parts, and their removal answer the indications, though such cases are, doubtless, rare. Usually it is necessary to proceed much further, often to remove, in connection with the cicatrix, surrounding diseased tissue in the brain substances. Horsley gave the direction toward this more radical treatment of brain lesions, which gives the promise of more favorable results.

²² Von Bergman. Surgical Treatment of Diseases of the Brain.

²³ Ibid.

Those cases seem more favorable for a complete cure where the epilepsy is of the Jacksonian type. There is a greater probability in such cases that the lesion is circumscribed that it can be found, and that the epilepsy has not yet become fully engrafted in the nervous system, to retain its existence even after its original cause is removed. Probably it is because of the latter condition that the convulsions so often continue to appear, notwithstanding the operation; especially may we believe this to be true when the convulsions have been of the type of ordinary epilepsy.

For the same reason there is much more probability of success if the operation be performed soon after the injury than when performed at a late period.

For non-traumatic epilepsy, trephining should only be resorted to in cases of the Jacksonian type. In these cases it may be taken for granted that a lesion, whether demonstrable or not, is in the motor area of the member wherein the spasm begins. It is, therefore, theoretically probable that the removal of this lesion may cure the disease. A number of such operations have been made.

Horsley²⁴ removed the facial centre in a boy of ten, somewhat demented, having from three to six epileptiform attacks daily, the attacks beginning at the left angle of the mouth, with some improvement as to the frequency of the attacks. Lloyd and Deaver²⁵ removed part of the arm and face centre in a man of thirty-five, with frequent daily attacks, beginning in the left index finger. The patient remained under observation nine months, during which time he had about ten convulsive seizures. Keen,²⁶ Naudcrede,²⁷ and Von Bergman,²⁸ also removed cortical centres for the cure of epileptic or epileptiform seizures, with "signal" symptoms pointing to the extirpated centres as the

²⁴ Brit. Med. Jour., Apr. 23, 1887.

²⁵ Jour. of Nerv. and Ment. Disease, June, 1889.

²⁶ Amer. Jour. of Med. Sciences, 1888, p. 452, and Wood's Reference Handbook, viii., p. 222.

²⁷ Med. News, 1888, No. 24.

²⁸ Surgical Treatment of Disease of the Brain.

seat of disease ; but in all these cases the epilepsy had more or less the character of traumatic epilepsy. In Keen's and Von Bergmann's cases there was some favorable modification of the disease, though the attacks continued to occur, with lessened frequency. Naucrede's case was only under observation three weeks after the operation, during which time no convulsion occurred.

In all these cases, except Von Bergman's, just before the removal of the cortical centre, its location was ascertained by applications of the faradic current. In all the cases a corresponding paresis or paralysis followed the removal of the centre, but, with the lapse of time, disappeared to greater or less extent.

It is yet too early to say whether such operations are to have a future. In the cases operated upon not all was accomplished that had been hoped for, and the possibility of doing harm cannot be overlooked.

ANÆSTHESIAS OF HYSTERIA.

This subject is considered by Dr. C. L. Dana in the "American Journal of Medical Sciences" for October, 1890. The characteristics of hysterical anæsthesia are :

I. Its frequent presence in the retinal field, and its peculiar distribution here.

II. Its distribution on the skin, affecting first the pain nerves, and its modification, disappearance, or transfer by metals, or suggestion, or cutaneous irritants.

III. Its peculiar involvement of the auditory nerve, causing deafness in high and even low tones, and dulling the hearing generally.

IV. The rarity of muscular and articular anæsthesia, except in connection with profound paralysis.

V. The involvement of the taste and smell.

The thirteen cases recorded well illustrate the four points in regard to hysteria that modern research has tended to bring out and emphasize :

The comparative frequency of the disease in men ; the common characteristics of the disease, whether caused by shock or trauma, local irritations, or general depressing influences ; the presence of some of the objective symptoms or stigmata of the disease in all cases ; and the combination of true hysteria with organic disease. L. F. B.

Periscope.

By WM. M. LESZYNSKY, M.D

THE RESISTANCE OF THE HUMAN BODY IN ITS RELATION TO THE FARADIC CURRENT.

In the "Deutsch. Archiv. f. Klin. Med." Bd. xlv., Hft., 304, L. Mann has undertaken the study of a question in electro-diagnosis, which, up to this time, has not been explained. That is: In what measure is the intensity of an induced current flowing through the human body influenced by the resistance of the latter? As it appeared in the course of his experiments that metallic or fluid rheostats weakened in a very high degree the faradic current, and thus greatly changed the result of the faradic examination, the author substituted body-resistance in place of metallic resistance. The experiments were arranged according to the three following methods:

1. The resistance was increased by the introduction of a second person in the circuit.
2. The resistance was modified by changing the size of the electrodes.
3. The resistance was diminished by the action of the constant current, and by various chemical agents applied to the skin.

The results of these experiments may be thus summarized: That the body-resistance exercises a much smaller influence upon the intensity of the faradic current than upon that of the galvanic; therefore the resistance during the faradic examination may be neglected. In order to explain this fact, another experiment was made.

Starting from the established principle that the epidermis forms the main resistance to the galvanic current, a dead body was utilized in place of the living second person mentioned in the first series of experiments. The skin was then removed at the points of contact with the electrodes, and the behavior of the body-resistance to both currents tested with intact and removed epidermis. The result was that the resistance of the skinned body affected in the same degree the intensity of the faradic as that of the galvanic current. As in the intact skin the body-resistance affects the faradic current so much less than the galvanic, the conclusion is that this differing behavior has its cause in the epidermis; or, in other words, that the same possesses no influence over the induction current.

As the neglect of the body-resistance to the induction current represents only an insignificant source of error, it follows that the simple measurement of the distance between the coils, without considering the resistance to conduction, perfectly suffices, for all practical purposes, for forming an estimate of the faradic irritability.

THE EXPERIMENTAL STUDY OF REFLEX PUPILLARY IMMOBILITY.

F. Mendel ("Deutsch. med. Wochenschrift," 1889, No. 47.) reports that in a number of dogs, cats and rabbits, shortly after birth, as soon as the eyes were open, the iris was in greater part removed by means of a single or repeated iridectomy. The majority of the eyes operated upon were destroyed through suppuration of the globe.

After several months the animals were killed, and the brains examined by a series of sections.

Atrophy of the anterior corpora quadrigemina and the corp. geniculat. ext., on the opposite side, and atrophy of the ganglion habenulæ on the side of the operation, was found in those cases with suppuration of the globe and optic atrophy.

As a rule, in those cases where the eyeball was preserved, atrophy of the ganglion habenulæ appeared upon the side of the operation.

The assumption, therefore, seems warranted, that in the ganglion habenulæ we have a centre for pupillary movement, whose development is arrested when it has no function to perform, as is seen in the case of iridectomized animals where no iris movement takes place. Darkschewitch has shown by means of anatomical and physiological studies, that the pupillary fibres of the optic tract enter into the pineal gland and into the ganglion habenulæ.

In consequence of this, the ganglion habenulæ would have to be looked upon as the reflex centre for pupillary movement.

Gudden's statement as to a prominence situated in front of the superior corpora quadrigemina; also the physiological researches of Bechterew, Hensen, Völkers and Christiani, all of whom emphasize the importance of the third ventricle, especially its posterior wall, in its relation to pupillary movement, can easily be reconciled with the results of Mendel's investigations.

What, then, is the course of the reflex arc?

Between the ganglia habenulæ there is a commissure radiating into the lowest part of the posterior commissure. The portion of this commissure adjacent to the atrophied ganglion habenulæ also becomes atrophic.

It can be traced for some distance into the posterior commissure, and according to M., indicates the direction of the sphincter nucleus.

But no atrophy was found among the collections of cells belonging to the oculo-motorius nucleus.

On the other hand, atrophy of Gudden's nucleus could be demonstrated in two cases. The reflex path for the rays of light which reach the eye, and the subsequent contraction of the pupil would have the following course: Optic nerve (optic tract) to the ganglion habenulæ without decussation, thence to the posterior commissure to Gudden's nucleus, and from the latter to the fibres of the oculo-motorius crest.

These researches indicate the method to be adopted in future in all anatomico-pathological investigations of this nature.

TRAUMATIC TABES.

In the "Zeitschrift f. klin. Medicin," Bd. xvii., hft. 1 u 2, Klemperer has tabulated the literature upon this subject. In all there were thirty cases, to which the author adds four new cases under Leyden's observation.

In a monograph entitled "The Gray Degeneration of the Posterior Columns of the Spinal Cord," published in 1863, Leyden was the first to describe in detail a case of traumatic tabes.

K.'s *first* patient was a locksmith, who was injured in the right leg from the fall of a heavy weight. The wound healed perfectly, yet, after a short time, the patient noticed that the right leg was weaker than the left, and that the scar became painful when he worked. Despite all treatment, the pain grew worse without previous exertion, and radiated over the entire leg. Towards the end of the same year, about eight months after the injury, the left leg became weak, and the pains, which were of a shooting character, affected both legs.

Not quite a year and a half after the injury, he was obliged to visit the clinic, and the diagnosis was made of well advanced tabes.

The *second* patient was injured during the late war by his wounded horse falling upon him. The right foot was crushed in the region of the ankle-joint, without fracture, but with extensive laceration of the soft parts.

The course of the disease was the same as in the last case, but not so rapid.

A *third* case originated as the result of a gunshot wound of the left leg. Painful spasm in the left calf muscles occurred after forcible exertion. Tabetic symptoms appeared slowly.

The *fourth* case had its starting point in a fracture of both clavicles, through his horse falling upon him.

All of these cases serve to confirm the view that tabes may arise as a result of peripheric injuries.

MUSCULAR ATROPHY AND CHANGES IN ELECTRICAL REACTION OCCURRING AS A RESULT OF CEREBRAL DISEASE.

Wasting of the muscles has been frequently observed in limbs paralyzed from brain disease, and in part accounted for by the existence in the cortex of trophic centres for the muscles. Considerable diminution in the electrical irritability has also been noted in several cases of cerebral hemiplegia and hemianæsthesia. However, in no case were qualitative changes present, either in the form of alteration in the galvanic formula, or slow contraction upon faradic or galvanic excitation.

Eisenlohr (*Neurologisches Centralblatt*, 1890, No. 1) reports a number of observations with autopsies bearing upon this point.

The *first* case was a female with aphasia, reported in the *Deutsch. Med. Wochenschrift*, 1889. In this instance there was atrophy of the small muscles of the right hand. There were also well-marked slow contractions in the abductor minimi digiti to cathodic closure and anodic closure. The autopsy revealed two abscesses about the size of a walnut in the left cerebral hemisphere. One was situated in the posterior part of the caudate nucleus, encroaching upon adjacent portions of the lenticular nucleus, corona radiata and internal capsule, and the other within the tegmentum of the crus in the subthalamic region and the posterior part of the optic thalamus. There was moderate secondary degeneration in the left pyramid and in the right lateral column of the cord. The anterior horns in the cervical and upper dorsal region were perfectly normal.

The *second* case occurred in a coppersmith twenty-one years of age, with epileptiform convulsions and weakness of the right hand, which ultimately advanced to paralysis of the extensors, paresis of the flexors and flaccidity of the muscles of the arm and forearm.

Upon electrical examination it was discovered that An CC was slower than normal in the right thenar and hypothenar group. As the disease advanced, convulsions occurred in the right arm, and subsequently the facial nerve and right leg became involved.

The faradic reaction was found diminished in the ball of the thumb and in the interossei. There was increased galvanic excitability in the first interosseus (active contraction), and in the thenar group changes in the character of the contractions were noted, *i. e.*, tonic contraction with An > C Ca C. The diagnosis was made of an abscess in the middle portion of a central convolution in the left hemisphere. Two unsuccessful attempts were made to evacuate the abscess.

The autopsy showed an abscess about the size of an apple in the left pre-central convolution. The microscopical examination of the hardened cord gave an absolutely negative result. No pathological changes were found in the ulnar nerve or affected muscles. It is worthy of note, that in both of these cases the anterior gray matter was found intact. In the second case neither the pyramidal tract nor the anterior roots were involved. Babinski assumed the existence of a "dynamic lesion" of the anterior ganglion cells in three similar cases of muscular atrophy from cerebral paralysis. Eisenlohr does not concur in this opinion, and is unwilling to class these galvanic changes with the genuine R.D. But he inclines to the belief that the trophic influences originating in certain portions of the brain can manifest themselves in a peculiar change in the muscular contractility, which, in its turn, causes special electrical reactions without any tangible histological changes taking place.

In a *third case*, in which a large area of softening was supposed to be present in the right hemisphere, there was decided diminution of the direct faradic reaction in several small muscles of the hand, and slighter in the arm, forearm and thigh muscles. In addition there were indications of qualitative changes upon direct faradic and galvanic excitation in several muscles of the hand (thenar, hypothenar, first interosseous), and marked diminution of nerve irritability in the paralyzed left upper extremity, in conjunction with conspicuous atrophy of numerous muscles and groups of muscles.

In a later stage several muscle groups in the hand and leg upon the healthy side showed beginning wasting. Eisenlohr believes that here we must assume the existence of

either a trophic influence passing from the cerebral centres of the right hemisphere to the musculature of the body on the same side, or the extension of a secondary degeneration of the direct and crossed pyramidal tracts into the gray substance of both anterior horns. Perhaps the atrophy from disuse may help to explain why the small muscles of the hand are so frequently affected.

GLOSSO-LABIAL-LARYNGEAL PARALYSIS.

In the *Gaz. des hopitaux*, 1889, No. 141, Moty reports the history of three cases of bulbar paralysis whose etiology is deserving of attention and consideration. All of those affected were members of one and the same family, whose parents were first cousins on the paternal side. The parents were in good health. Among others of the family several marriages of blood-relations had taken place, without any apparent ill effect upon their progeny. In the above-mentioned family there were eleven children. The eldest was twenty-seven years of age, in good health, married, and the mother of two living children. The second child, also a daughter, twenty-five years of age, was healthy until her eighth year, when the symptoms of bulbar paralysis began.

In the third daughter, twenty-three years old, the disease began to develop itself at the seventh year. The fourth child, a son of twenty-seven years, was healthy at birth, but through a fall in his first year he received a dislocation of the right thigh. The first signs of the disease appeared likewise in the seventh year. The fifth child is a son nineteen years of age. Three years ago he acquired a tuberculous ostitis in one of the thighs. The sixth, seventh, eighth and ninth children were boys. The tenth and eleventh were girls. The last one was born five years ago. The author, who doubts the legitimacy of the first child, calls attention to the fact that the disease affected the eldest three legitimate children, and according to the general laws of heredity, the first children suffered most from hereditary influences. Besides the relationship of the parents, there was also the pernicious influence of alcoholic excesses.

RELIEF FROM A GRAVE TYPE OF EPILEPSY FOLLOWING THE CORRECTION OF AN EXAGGERATED CONVERGENT STRABISMUS.

Dr. Chas. H. Beard (*Archives of Ophthalmology*, Apr. and July, 1890), reports the case of a German barber fifty-two years of age, who suffered from excruciating pain in

the left eye, affecting the distribution of the fifth nerve on the same side. This pain, which began in the eye, had been first felt eight or ten years previous, and had become almost constant. At about the same time the left eye deviated inwards, the pain and the convergence gradually increasing, until the one had become unbearable, and the other had progressed to the extreme limit. It was only by a decided effort at abduction that a portion of the cornea could be brought into view. Four or five years previous he began to have epileptic attacks, which increased in frequency and severity until he had as many as three or four fits within twenty-four hours.

The mental faculties were not affected, in fact the man was intellectually much above the average of his class. There was also in this case total deafness, evidently due to disease of the labyrinth which had begun twenty-five years before. Otherwise he enjoyed excellent health.

The morbid eye had grown amblyopic, the retina retaining its function but feebly on the nasal side only. In all respects not mentioned, the eye was normal. Vision in the fellow eye was good, and there was no error in the refraction of either eye.

The operation of advancement of the left external rectus combined with a thorough tenotomy of the left internus, was done under ether. Recovery was prompt, and the result most gratifying; for not only was there correction of the deformity, but from that time till the patient's death, which occurred from a railway accident on the 22d day of June, 1889, fully fifteen months later, he neither suffered from the old agonizing pain, nor had a single fit of epilepsy.

THOMSEN'S DISEASE (*Myotonia Congenita*).

Erb (*Deutsch. Archiv. für Klin. Med.* Bd. XIV., Hft. 5 u 6), briefly relates the history of the cases of Thomsen's disease published since his first contribution in 1886.

There were seven cases in all. While other observers have confirmed in almost every detail the symptoms described by E., it is remarkable that none of these could discover the rythmical wave-like contractions manifested under the stabile influence of the galvanic current.

As these peculiar contractions were demonstrable in his five recent cases, he considers them as a constant, and therefore probably pathognomonic symptom. The fact that others have not succeeded in evoking this symptom he believes must be accounted for by the circumstance that on the one hand it has not been searched for with the nec-

essary perseverance, and on the other, that the examiners have not employed all the means and artificial devices which, in the more difficult cases, are calculated to bring to view the phenomenon in question. Therefore in the published histories of cases he again refers to these methods, that have already been explained in his earlier monograph. The five new cases are typical pictures of the disease.

In a supplement he relates four additional cases from three other observers published since the completion of his work, in all of which the last-mentioned phenomenon was elicited. Two patients showed, in addition, the interesting feature that the effect of cold exercised a pernicious influence over the course of the disease.

By LOUISE FISKE-BRYSON, M.D.

A NEW MEDICAL JOURNAL.

"The Baltimore Medical and Surgical Record," containing forty pages, including a Berlin letter, a portrait of Dr. H. P. C. Wilson, and a variety of minor items, began its existence in Baltimore, October 1. The editor and proprietor, T. H. Graham, proposes to call to his aid specialists in the various departments, who are expected to contribute original articles, editorials, book reviews, and miscellany, for which they are to be duly compensated. The first number is in every way creditable.

PHYSICAL AND MENTAL TRAITS OF CRIMINALS.

Mr. Havelock Ellis has embodied in convenient form, in one of the valuable books of the year, called "The Criminal," all the latest words that have been spoken in different countries concerning this interesting degenerate. The following refers solely to the instinctive criminal, the one who is born, not made; and has nothing whatever to do with the accidental criminal made such by pressing necessity, such as impending starvation, by insanity, by passion, or by accident. The typical malefactor often learns how to work for the first time in prison. His chief object in life—the whole art of crime—lies in the endeavor to avoid the necessity of labor. He has now been measured, weighed, subjected to all known tests, and studied from the standpoint of structure, function, and chemistry. The criminal is not like other men,—not more, but infinitely less, resem-

bling in many points the idiot and the insane. Lombroso, the author of "*L'Uomo Delinquente*," says that "in general, born criminals have projecting ears, thick hair, enormous jaws, a square and projecting chin, large cheek bones, and frequent gesticulation." Well-shaped heads are seen, but beauty of countenance never. The senses are all dulled, except that of sight, which is abnormally acute, like that of imbeciles. Insensibility to pain is a marked characteristic. Extraordinary agility, sometimes facilitated by unusual length of arm, is a characteristic that the criminal shares with the lower races of mankind and with apes. Left-handedness frequently exists, and inability to blush, as among many savages and idiots. Abnormities of tendon reflex and sexual anomalies are frequent. In regard to intellect, the criminal is astute rather than intelligent, astuteness being a natural, instinctive quality incapable of cultivation, and common to animals, savages, many women, imbeciles, and precociously perfected in children. It deals exclusively with concrete cases. Intelligence, on the other hand, develops slowly even among the highly endowed, is a quality of infinite variety, dealing with language and abstract questions, and requires training for its full perfection.

CEREBRAL LOCALIZATION.

Dr. Ferrier's fourth Croonian lecture appears in the "*British Medical Journal*" of June 28, 1890. It treats of the auditory centre. The facts of human pathology support the view that the sense of hearing is localized in the temporal lobe, more especially in the superior temporal gyrus.

LANDRY'S PARALYSIS.

There is a case of acute ascending paralysis reported by Dr. Godfrey Carter in the "*British Medical Journal*," May 17, 1890. Owing to the onset, symptoms, and family and personal history, Dr. Carter suggests that Landry's paralysis may be due to chill, causing an effusion of fluid in the central canal of the spinal cord, this fluid rising higher and higher and exerting most pressure where the column of it is deepest and heaviest—that is, from below upwards—until finally the ventricle of the medulla is reached.

HYSTERICAL PSEUDO-TABES.

In the "*Gazette Médicale de Paris*," Dr. Pittes has a paper on this subject. Hysterical pseudo tabes is the name given to cases that present motor and sensory symptoms, giving

more or less faithfully the syndrome of locomotor ataxia without the medullary lesions. Sometimes there is cerebellar trouble or irregular spinal sclerosis, or diffuse alterations in peripheral nerves. But more often the condition is purely neuropathic, without any changes in nerve centres or peripheral nerves. There is also a neurasthenic ataxia. In both the symptoms—even to the Romburg-Brauch—are like those of true ataxia, to which are added hysterical stigmata, such as narrowing of the visual field, abolition of the pharyngeal reflex, and hemi-hypoesthesia; and in the neurasthenic form, the peculiar headache and anxious melancholy of neurasthenia. The diagnostic difference between a false and true ataxia rests upon the condition of the patellar reflexes, the Argyle-Robertson pupil, and the optic neuritis and atrophy, together with Westphall's symptom and the mode of onset.

ARTERO-SCLEROTIC VERTIGO.

Grasset points out, according to the "*Gazette Hebdomadaire de Médecine et de Chirurgie*," that cardio-vascular vertigo is an important symptom in the initial stage of arterial sclerosis, and is probably due to transient constrictions of arterioles of the bulbe. It presents these types or degrees of intensity: simple vertigo, vertigo with epileptiform crises, vertigo with slow pulse, and syncope or epileptiform seizures. It is an indication for the treatment of arterio-sclerosis, for which the most efficacious remedies are iodide of sodium and trinitrinc.

SALAAM CURED BY MASSAGE.

The "*Gazette Hebdomadaire de Médecine et de Chirurgie*," quoting from a Brazilian journal, records the cure of salaam in a child of five by massage of the cervico-dorsal portion of the spinal region. The head moved continually, with increasing intensity. The child was otherwise well, without defective personal or family history.

VIVISECTIONS ON THE HUMAN BRAIN.

Dr. Brown-Séquard, in the "*Archives de Physiologie Normale et Pathologique*," for October, 1890, considers that various recent surgical operations performed according to the new doctrines of cerebral localization have established facts contrary to the principles of these doctrines in great degree. From an observation of twenty cases, the author arrives at the conclusion that the parts of the brain thought

to be the seat of psycho-motor centres do not possess any such function. If they cause paralysis, it is by means of some irritation transmitted to them through other nerve-centres.

SUSPENSION AND POSTURAL METHODS IN NERVOUS DISEASE.

Dr. Allan McLane Hamilton, in the "Medical Record," August 30, 1890, treats of this. Irregular cases of ataxia are those most benefited by suspension, and functional and irregular conditions, as traumatic hysteria, more or less diffused neuritis of doubtful or unknown origin. An analysis of five carefully observed cases of ataxia treated by suspension is given, and an account of Hessig's method of spinal extension, together with a description of the author's new apparatus.

RESTRICTION OF THE FIELD OF VISION IN SYRINGOMELIA.

In "Médecine Moderne," August 28, 1890, Dr. Dejerine and Dr. Tuiant have an exhaustive paper upon this subject. Up to the present time, except in optic nerve, retinal, and brain trouble, restriction of the field of vision has only been found in hysteria proper, in traumatic hysteria, and traumatic neuroses. In epilepsy, narrowing of the field is not permanent, existing only immediately after the attack. That changes in the visual field are always present in syringomelia, the authors are not prepared to state. It did exist in the nine patients examined. These exhibited no symptoms whatever of hysteria, and had at no time sustained an injury.

SENILE HYSTERIA.

Dr. Maurice de Fleury, in "Médecine Moderne," September 4, 1890, states that in this condition it is the easiest thing to make a mistake in diagnosis. Every symptom of narrowing of the œsophagus may be present, and this suggests a cancer. An attack like angina pectoris turns the attention to atheroma of the aorta and coronary arteries. Dyspnœa gives the idea of beginning pneumonia, congestion, or pleurisy. And the lymphadenitis, painful constipation, hepatalgia, uterine colic, burning about the meatus may also mislead. Senile hysteria differs from that of adult life in that it presents the minimum of motor and sensory phenomena that are under central control, and the maximum of painful and spasmodic symptoms under the influence of the sympathetic.

EXCISION OF THE CEREBRAL CORTEX AS SURGICAL
TREATMENT OF PSYCHOSES.

The "Gazette des Hôpitaux," August 21, 1890, notes Dr. Burckhardt's paper on this subject before the International Congress in Berlin. The author views psychoses as due to disseminated lesions in which the point of departure varies considerably. According to his idea, it is permissible to remove such portions of the cortex as may be considered the starting point of psychic disturbances, and to destroy such associated fibres as bring about pathological conditions. The results of operating upon six patients have been most satisfactory.

LESIONS IN GENERAL PARESIS HISTOLOGICALLY
CONSIDERED.

In "La Médecine Moderne," September 11, 1890, Klippel gives interesting data upon the minute changes in general paresis. The ganglionic cells show degenerative changes. In the medium-sized arterioles the most pronounced lesions are found. In the light of minute morbid anatomy, the investigator asks whether general paresis starts as a disease of the brain, cord, and peripheral nerves, or as a disorder of visceral organs, since these always present lesions often as profound as those of the nervous system. The changes in the kidneys are the most constant and the most profound. Sometimes the kidneys are small, hard and sclerotic. More often they are pale and soft, showing evidence of fatty degeneration. The evidences of degeneration in the parenchyma are as marked as those of the brain. The liver also shows fatty degeneration. Sometimes the heart is sclerosed; sometimes in a state of pigmentary fatty atrophy. The condition of the lungs is almost always the same: broncho-pneumonia nearly always exists on the inferior portion of one side. Intense congestion may mask disseminated hepatization that the microscope reveals. The pulmonary capillaries are also greatly congested. Congestion and desquamative catarrh are habitually present, which appear more as the spleno-pneumonia of marasmus rather than as a part of the original and primitive disease. It would appear from Klippel's investigations that general paresis is a degenerative process, in which the inflammation, meningitis, sclerosis, etc., are secondary to the destruction of the cells and nerve fibres.

VISUAL DISTURBANCES IN TABES DORSALIS, AND AN ATTEMPT AT A COMPREHENSIVE EXPLANATION OF THE SYMPTOM-COMPLEX OF TABES.

Dr. E. Berger's remarkable paper upon this subject appears in English dress in the "*Archives of Ophthalmology*" (October, 1890), translated by Dr. Ward A. Holden. The symptoms of tabes, besides those caused by disease of the spinal cord, consist of disturbed function of special cerebral nerves, sometimes of the cerebrum and probably of the cerebellum.

Of the general disturbances of the brain should be mentioned headache, disturbance of speech, failure of memory, and disturbances of intellect; yet these are unusual. More frequently cerebral nerves are affected—the optic nerve, the nerves of the ocular muscles, and single branches of the fifth. It is seldom that the hypoglossal, the glosso-pharyngeal and the olfactory nerves are also affected. Occurrences of diabetes mellitus and insipidus, of bronchial asthma, of nephralgic crises, and of rectal and urethral colic are reported.

Pathological changes are found in the medulla, in peripheral, cerebral and spinal nerves, in the spinal ganglia, and in the sympathetic nerve. Disease of the optic nerve appears first in the periphery, and affects the central fibres later. The nerve fibre and ganglionic cell layers become atrophic, owing to increase of connective tissue, that begins with a formation of fat drops and an increase of pigment (Leber, Raymond, Arthaud). The author thinks that his own experiments on animals would confirm the idea that disease of the medulla can explain the symptoms of tabes, as here may be found the connecting link between the spinal and the optic nerve affection.

The analysis of one hundred and nine cases shows that tabes appears most frequently during the most vigorous period of life—between the ages of thirty and forty-five years. It has appeared as early as the age of fifteen, and as late as seventy. The cases occurring at the earliest and latest periods of life are free from severe complications. These occur during the period that is normally of greatest activity. Of the cases studied, there were forty-four men with syphilitic history and forty-nine non-syphilitic; among the women, three syphilitic and thirteen free from specific taint. On an average, the first symptoms of tabes appeared eleven and a half years after the primary affection of syphilis.

Tabes in syphilitic cases begins for the most part with cerebral symptoms, while in the non-syphilitic the spinal symptoms manifest themselves earliest.

There is often a slight narrowing of the palpebral fissure, even when no paralysis of the oculo-motor nerve exists, and epiphora is about half the cases, together with fibrillary twitching on closing the lids, and a diminished intra-ocular tension to the extent of causing the ball to be quite soft. Inequality of the pupil often exists. An irregularity, however, hitherto unnoticed, is in the shape of the pupil. It is often elliptical, with its long diameter most frequently oblique from downward and outward to upward and inward, though the long diameter may lie horizontally or in some other direction. Their action to light is lost first; later the reaction to accommodation. This peculiarity is found also in progressive paralysis; otherwise it is rare. Stolzenberg found it in many cases of cerebral syphilis. Bisulphide-of-carbon poisoning has produced it (Uhthoff). Mauthner's explanation of pupillary immobility, verified by the experiments of Moeli and Knapp, is, that the fibres for the pupillary light reflex, lying in the wall of the third ventricle, suffer from a chronic ependymitis and a consequent sclerosis.

Cases with paralysis of ocular muscles are more disposed to optic atrophy than those in whom this paralysis is absent. The left eye is first affected twice as often as the right. The greater number of atrophies develop in the *stad. prætact.* If this stage is once passed, the danger of its occurrence is less. Should an optic atrophy develop later, the consequent blindness follows more rapidly.

Erb gives the following as the usual order for the loss of color-perceptions: (1) green, (2) red, (3) yellow, (4) blue. No relation exists between the contraction of the visual field, the color-fields, the disturbance of central vision, and of color-perception.

With regard to the blood-vessels of the eye, the capillaries must be dilated, at least at the beginning of the atrophy. The central vessels undergo the following changes: (1) arteries and veins normal; (2) arteries narrow, veins normal; (3) arteries narrow, veins dilated; (4) arteries and veins narrow. The two groups of functional visual disturbances, apart from dioptric errors, are: (1) those in which the fault lies in nerve-fibre conduction, and (2) those in which the functional error is caused by a disturbance in nutrition. The causes of the visual disturbances must be sought for in the vessels alone.

Many facts point toward vaso-motor disturbance from central causes. In regard to the treatment of tabetic optic atrophy, mercury is positively harmful. If injections of strychnine do no good, rest in a darkened room and iodide of potassium are in order. Among syphilitic patients there is greater tendency to permanent and multiple paralyses of ocular muscles than among non-syphilitic. Paralysis in the regions supplied by the facial nerve also occurs.

Thus it will be seen that many of the symptoms of tabes may be explained by a lesion of the nerve-nuclei in the fossa rhomboidalis and its continuation into the aqueduct of Sylvius. The difficulty in closing the lids is probably due to a slight functional disturbance of the upper nucleus of the facial nerve, which is in relation with the nucleus of the fifth nerve; the myosis, to paralysis of vaso-constrictors in the medulla; the reflex iridoplegia, to a lesion of the fibres running in the wall of the third ventricle to the nucleus of the sphincter pupillæ; and the diminished intra-ocular tension—existing in one-third of the cases—to lowering of the tone of the vessels. Lachrymation in tabetic patients is a vaso-motor neurosis, analogous to the unilateral hyperidrosis that is sometimes present. The sympathetic nerve is not the cause of certain ocular symptoms, but the medium by which these are carried from the central nervous system to the eye. A number of nerves whose nuclei lie in the medulla may become affected. The whole facial nerve, or only its lower twigs, may become paralyzed. The auditory, the vagus, the fifth, and rarely the spinal accessory may suffer functionally, and, according to some authorities, the glosso-pharyngeal and hypoglossal nerves as well. In general, the nerve-nuclei in the medulla nearest the middle line suffer more frequently than those further outward, and the nuclei in the upper portion more frequently than those lower down. An affection of the medulla is indicated by the development of diabetes mellitus in tabes, and by febrile symptoms. The author then states that the following points are to be considered:

1. Whether we are justified in assuming the vascular centres to be located in the medulla.
2. That, in consequence of disease of the nerve centres, vascular changes can develop.
3. That vascular changes occur in the various nerve regions which are the seat of the morbid process in tabes.
4. That the vascular changes are also the cause of the disease of the nerve-tissue.

5. Whether trophic nerves cannot cause simultaneously the changes in the affected nerve regions.

A careful survey of these important considerations seem to the author to support his view of *tabes dorsalis*.

THE EFFECT UPON THE BODILY TEMPERATURE OF LESIONS OF THE CORPUS STRIATUM AND OPTIC THALAMUS.

W. Hale White, in the January number of the "*Journal of Physiology*," has a paper with this title. Hitherto, in experiments upon animals, but little attention has been paid to the normal temperature of the animals operated upon, and but little to the effect of the anæsthetic or the trephining apart from the injury. The author's experiments lead him to believe that the corpus striatum and optic thalamus have, in rabbits at least, the power of modifying the temperature of the body, and that the surrounding white matter has no such power.

KNEE-JERK AND ITS PHYSIOLOGICAL MODIFICATIONS.

The "*Journal of Physiology*," January, 1890, contains a series of interesting experiments that demonstrated the changes in knee-jerk from purely physiological causes. Exaggerated reflex can be inhibited by the stimulation of the skin or peripheral nerves (Nothnagel, Lewinski, Erb.); but the reflexes thus inhibited were in nearly all the cases reported the clonic movements produced under morbid conditions, by stretching the tendons of the muscles concerned. Erb mentions a case of myelitis from compression in which tendon reflex and trembling of the legs was stopped at once by pinching the skin of the abdomen. If the patient clinches the hand when the patellar tendon is struck, or makes other violent movement, the coincident jerk is increased (Jendrassik). Volitional acts directed to other parts of the body, painful stimulation of the nerves of the skin by pinching, by the application of heat, cold, or electricity, exposing the eyes to the light of burning magnesium wire, will increase knee-jerk (Mitchell and Lewis). Fatigue, hunger, enervating weather, sleep—conditions which decrease the whole activity of the whole central nervous system—decrease the average patellar tendon reflex. Rest, nourishment, invigorating weather, and wakefulness increase it (Lombard).

TINNITUS AURIUM.

The "British Medical Journal," September 20, 1890, has a paper by Dr. H. Macnaughton Jones, in which a table of two hundred cases of tinnitus aurium is given, accompanied or caused by various abnormal conditions. Connections exist between the auditory nuclei in the medulla and pons and the cortical centres on either side of the brain. According to Ferrier, the sense of hearing is in greater part situated in the temporal lobe, and more especially in the superior temporal gyrus of this lobe. Tinnitus may occur in both ears as the result of unilateral irritation, the effect of the intimate connection of the auditory nerves and centres. Baginsky says that the auditory nerve is in relation with the auditory centre of the cortex through the lower fillet of the opposite side, and thence by means of the posterior tubercle of the corpora quadrigemina and internal geniculate body to the medullary fibres of the cortex. The receptive auditory centre is, possibly, in part situated in the cerebellum. An irritation occurring either in the labyrinth, the auditory nerve, or receptive centre, (Kendrick) will produce a stimulus in the hearing centre which will cause a sensation or series of sensations which will always be referred to the labyrinth. Such irritations may be caused by anything that interferes with the normal equilibration of the fluid in the labyrinth, by direct or reflected irritation of the nerves of the external meatus, the middle ear or labyrinth. They may follow or attend on the presence of abnormal constituents in the blood which circulates in the receptive centre, as well as alterations in the blood supply due to increase or diminution of blood pressure. This latter condition, occurring either at the periphery, in the cerebellum, or in the course of the auditory nerve, would be in itself sufficient to start an irritation of the hearing centre.

A NOTE.

Dr. E. H. Van Deusen's generous municipal gift to Kalamazoo carried with it the proviso that there should be a set of rooms set aside for the exclusive use of the Kalamazoo Academy of Medicine.

NEW YORK NEUROLOGICAL SOCIETY.

Meeting, November 4, 1890.

The President, DR. LANDON CARTER GRAY, in the chair.

ASTASIA AND ABASIA.

A patient was presented by Dr. G. M. HAMMOND exhibiting the symptoms laid down as characteristic of this unusual affection. The patient, a young woman, had never been the subject of any serious illness excepting Pott's disease, which had come on during childhood. Over a year ago the speaker had attended her through an attack of nervous prostration. During her illness she had suffered from aphonia; the difficulty in standing and walking was not discovered until she was able to leave her bed. On regaining her strength sufficiently to walk around it was observed that she invariably walked by first advancing the left leg and then drawing the right one up to it. When she attempted to walk naturally, immediately that the right foot touched the ground her body would revolve rapidly to the right, when, after making a revolution and a half, she would sink to the floor. Physical examination of the limbs revealed nothing abnormal. The patient while seated or lying down could move both legs normally; with the right leg, however, more mental effort was required to make the movements. The patellar tendon reflex was normal on both sides. There was no ankle clonus, no anæsthesia, hyperæsthesia, or any other disorder of sensibility in any part of the body with the exception of slight loss of the muscular sense in the right leg. The electrical reactions, both qualitative and quantitative, were normal. The field of vision and the color sense were found normal. The senses of hearing, touch, pain and temperature were tested without anything abnormal being discovered. There was some resistance to passive flexion and extension of the right leg. These symptoms then of difficulty in standing and of inco-ordination and ataxia of movement for the act of walking, but not for other muscular acts, corresponded accurately to that condition described by Bloeg under the title of astasia and abasia. Bloeg was of the opinion that astasia and abasia was a condition pathologically similar to agraphia. The speaker did not see anything in these cases to substantiate this view.

People afflicted with the disease under consideration could make the motions of walking perfectly well if they were allowed to lie down, but it had never been claimed that an individual suffering from agraphia could write any better in one position than he could in another. The condition, it seemed to the speaker, depended upon a loss of the power of adjusting muscular contractions so as to retain an exact equipoise or equilibrium. This was, of course, a defect of the muscular sense. There was no known tract in the spinal cord, disease of which would be followed by these symptoms. Bloeg had attempted to make a distinct neurosis of this class of cases, and claimed that a differential diagnosis between hysteria and astasia and abasia could readily be made. In the latter disease there were no hysterical stigmata, he claimed, no constant paralyses or constant contractures. But the latter were by no means characteristic of all cases of hysteria, and when it was considered that every case of astasia or abasia had been accompanied by some other symptom or symptoms, such as, for example, hyperæsthesia, anæsthesia, aphonia, contraction of the visual field and temporary color blindness, all of which frequently accompanied hysteria, and since the disease under consideration was purely functional in character, no gross or microscopical lesion ever having been discovered in them, it would not be difficult to believe that astasia and abasia was merely an uncommon type of an hysterical affection.

Dr. C. L. DANA said that if it were possible to exclude any organic trouble as a factor in the case there seemed nothing left but to give the condition the name which Dr. Hammond had done. It was by no means clear that this so-called disease deserved a separate clinical position, and they were by no means aware of all the vagaries of the trouble. He thought that the diagnosis might be accepted as a provisionally correct one.

Dr. LOUISE F. BRYSON said she had recently been reading a case reported in a French journal of what was known as "left and right-sided disease," in which the patient always had to walk to the right. Physiologically the muscles of the right side were stronger than those of the left, and perhaps the case was one of exaggerated function of the muscles of the right side.

Dr. G. W. JACOBY said that in a recent number of the *Berliner Klinische Wochenschrift* Dr. Binswanger had stated his belief that the whole trouble resulted from a physical condition, as the same phenomena were found in other men-

tal states. He had not seen a case' exactly like this, but others which reminded him very much of it. He was inclined to consider the condition as a psychic manifestation. Women, after long confinement in bed, would sometimes, when attempting to walk, find themselves too weak to do so, and immediately conclude that they had lost the power. While lying down or sitting they would have entire control of their limbs, but when they essayed walking, then came the fear. It was a psychic disturbance of equilibrium. He thought that Binswanger had done as much to clear away doubt in this class of cases as others had done to confuse.

The PRESIDENT said he had never seen anything like this case. The cases of hysterical paralysis which he had seen were typical forms of paraplegia. He had also read the two cases described by Russell Reynolds, who had called them "paralysis of idea." He did not think it was well to designate this case as one of hysteria on account of the presence of some spots of anæsthesia, because it had been shown that this occurred in a great many different nervous disorders, both functional and organic. It seemed better to accept the case as a clinical entity and hold any opinion in reserve as to the cause of the manifestations.

SYRINGO-MYELIA.

Dr. J. C. SHAW presented a patient to the society suffering with, as he had diagnosed, syringo-myelitis. The patient, a single woman, thirty years of age, had always good health until about six years ago, when a weakness of her left hand was noticed. This condition had steadily increased up to the present time. For the past three years she had had a constant aching in the left arm, shoulder, and side of the neck, and lately on that side of the head. For two years there had been a numb spot on the inner side of the left arm. She had constant sensations of burning on the left side of the face and neck, with flashes of heat and cold. There was a small spot on the back of the head where this burning sensation was greater than anywhere else. She presented an atrophy of the small muscles of the left hand, which had been in existence for six years, and was gradually growing worse. There was also slight atrophy in all the muscles of the arm, shoulder and face of the left side. In the area of the numb spot the tactile sensibility was impaired. The thermic sense was greatly diminished over the entire left side, and in the right lower extremity as

well. The reflexes were exaggerated. While the examination of the patient had not been as careful as it might have been, the speaker thought that it was sufficient for the purpose of making a diagnosis.

Dr. B. SACHS thought the personal equation was a powerful factor in this case. It certainly had been so that evening. The case did not seem to him to be one of syringomyelia. The atrophy was not marked enough, particularly about the shoulder. The sensory symptoms were not so distinct as in a typical case. So far as he could judge, the case seemed one of amyotrophic lateral sclerosis, though further examination or observation might lead him to a different conclusion.

Dr. A. M. STARR said that there were several features about this case which reminded him of one which had come under his observation. He had not brought these points out because he did not know that they belonged to syringomyelia. One of these peculiarities was the noise made in the throat—a sound as of alarm. This had been present in his patient, who was by no means an hysterical girl. He had regarded it as a muscular contraction of the larynx during inspiration. His patient would make the noise whether she was quietly conversing in his office or was before a class of students. This feature was to be taken into consideration. He thought that the stationary condition of the atrophy in this case indicated the existence of syringomyelia rather than that of amyotrophic lateral sclerosis. He had demonstrated pretty conclusively the changes in pain-sense by sticking the point of a needle in the patient's arm without her knowing it. There was no mistake about it, for he had put the needle in a quarter of an inch. Then there was the history of a loss of temperature-sense. The patient had noticed in putting her hands in hot water that there was a difference between the two sides. Therefore, bearing in mind the non-progressive condition of the atrophy, the existence of changes in the temperature and pain senses, he supposed they were warranted in making a diagnosis of syringomyelia at the present date.

Dr. SACHS thought the question depended upon the actual condition of the sensory derangements in this case, and of course the examination had been but cursory.

Dr. W. R. BIRDSALL thought that where the results of examination were so at variance, it would be hardly worth while to attempt any expression of opinion in the way of diagnosis. It had been his impression from the description of cases of syringomyelia of which he had read, and in

which an autopsy had been held as confirmatory evidence, that the histories had given the pain and temperature sense as having been both affected. He should say that the case before them was at least typical in this respect. As to this disease, it was a remarkable fact that, during the past year, of the cases in which syringo-myelia had been diagnosed during life there was no autopsy, while in those autopsies which had revealed the existence of the disease, its presence had not been suspected during life.

Dr. DANA said that last spring he had a patient in his hospital service who had presented many similar symptoms. There had been atrophy in the muscles supplied by the ulnar nerve and in the small muscles of the hand. There was also anæsthesia, involving the temperature and pain senses. The atrophy had slightly involved the opposite side. There was also a belt of anæsthesia over the lower portion of the trunk, and extending to the thighs. There was no disturbance to the sensory functions. The girl had gradually developed symptoms of bulbar paralysis without any sensory symptoms accompanying. He had been obliged to regard this as a typical case of progressive muscular atrophy. He had since seen a case of progressive muscular atrophy in which sensory symptoms were present. If the symptoms of bulbar paralysis were developed in the case before them, it would, he thought, turn out to be a case of progressive muscular atrophy. As to amyotrophic lateral sclerosis, it was simply another name for the same disease.

The PRESIDENT said the only way to make a diagnosis of syringo-myelia seemed to be to make an autopsy. The value of the loss of thermic sense in a patient as a diagnostic point was, to a great extent, vitiated by the fact that the relations of this sense to other organic spinal diseases were unknown. He thought they would be unable to establish the fact satisfactorily that this was a case of syringo-myelia until the woman died.

A CASE OF SPINA BIFIDA, WITH SUPPURATIVE MENINGITIS
AND EPENDYMITIS OF BACTERIAL ORIGIN.

By Drs. L. Emmett Holt and Ira Van Giesen. See page 774.

Dr. L. E. HOLT said he had seen one other of these cases of hydrocephalus in which the disease had existed without any symptoms during life. He thought there were probably a great many more than was usually supposed. He had been surprised to find that the ventricles contained

several ounces of fluid. He had seen several cases of basilar meningitis in which only a moderate amount of distention of the ventricles was found. In two of these cases the entire contents of the lateral ventricles would not have exceeded an ounce.

CEREBRAL COMPRESSION.

Dr. E. D. FISHER read a paper with this title. He said that while he had nothing new to present, he thought that he could settle definitely the question of the influence of compression on the cerebral mass within the skull, and whether the cerebral substance was, *per se*, compressible without interference with its capillary circulation or function. Bergnamis and Adam Knoiez (?) hold the views that the brain substance was incompressible, the only conditions of change possible in the cerebral volume being those dependent on the displacement or variation in the cerebro-spinal fluid or the cerebral circulation, these standing in converse relation to each other. The question of the compressibility of the brain depended on which of the elements comprising the brain was most liable to compression; as the blood pressure was higher than that of the cerebral fluids, it was possible that the tissue fluids were first affected. Much depended also as to whether we regarded the liquor cerebri as a secretion or as a transudation from the blood vessels, as in the latter case we would have to consider arterial tension as a very important factor in cerebral compression. The vascular centre was not only situated within the medulla, but probably also within the brain: *i. e.*, the corpus striatum or optic thalamus. The brain possessed a mechanism of its own for increasing its blood supply, independent of increased cardiac action. Experimentally it had been proven that cold acted deeply within the brain. Its good effect was very marked in the headaches of the anæmics, the ice-bag being an efficient remedy. The cold probably acted by increasing the blood current rapidity in the capillaries, and by causing spastic contraction of the arteries. In these cases, the amount of the blood passed through the brain by increasing the rapidity made up in quantity for the quality, thereby maintaining the nutrition. The extent of a cerebral hæmorrhage depended on the arterial pressure or tension, the intra-cerebral pressure, and also on the resistance of the brain substance, the latter, of course, depending on the site of the hæmorrhage. By spastic contraction of the arteries of the brain we really had

active hyperæmia, the decreased volume of skull contents causing increased capillary circulation. By paralytic dilatation of the arteries we had passive hyperæmia, which was, in fact, anæmia, the blood being no longer in a proper state to carry on the nutrition of the brain, as the increased volume of the brain caused retardation of the capillary circulation, and probably also interfered with the venous circulation. The speaker's experiments had been made by exposing the pia mater and observing the changes produced in the blood vessels. Extension of the sciatic nerve produced increased volume of the brain. Compression of the carotids caused marked loss of volume. Asphyxia caused expansion of the brain. Chloral caused anæmia, with marked contraction. Chloroform contracted, ether at first contracting and subsequently expanding the brain. Strychnia caused marked expansion, as did digitalis and small doses of alcohol. Caffeine and the acids caused expansion, while the alkalies produced the reverse result. Drawing from his experiments, the speaker concluded that the blood supply of the brain varied directly with the blood pressure in the systemic arteries, and that the extensibility of the walls of the cerebral vessels allowed of great variation in calibre. The vaso-constrictor centres were excited directly by disturbance with the nutrition of the nervous system, as in anæmia, asphyxia, and so forth. Finally, that the essential product of cerebral metabolism contained in the lymph spaces bathed the walls of the arterioles and could cause variations in the calibre of the vessels, that this mechanism reacted on the brain, and that by this means the vascular supply could be varied locally according to local varieties of functional activity.

Dr. JACOBY said that the demonstration that the nerve tissue could be compressed in its molecules and anatomical elements had been claimed by one author. Another had maintained the non-compressibility of these elements, but had further stated that the effect of hæmorrhage was due to anæmia of the brain. This was as a theory substantiated by comparing the clinical symptoms in such hæmorrhage with the symptoms caused by the injecting of lycopodium powder into the cerebral vessels, and producing thrombi. He then referred to the experiments of one who had demonstrated the displacement of the cerebral spinal fluid. In one case in which rice was injected the aqueduct of Sylvius was found to be ruptured, and the lateral ventricles flattened together. This experimenter had inferred that the very suddenness with which the compression was exercised

had caused the violent displacement of the cerebro-spinal fluid and consequent rupture. The same observer had also estimated very minutely the amount required to oppose the arterial tension, and had claimed that if at any time an effusion took place on the surface of the brain, to entirely balance this death must instantly result. Dr. Fisher had pointed out the necessity of maintaining arterial tension, rather than depressing it, upon this very theory. It had been suggested that in cerebral hemorrhage the head should be hung down to mechanically send blood to the head, and thus oppose the effusion which was taking place from the ruptured vessels. He did not know whether this had ever been put into practice.

PHILADELPHIA NEUROLOGICAL SOCIETY.

Stated Meeting, October 27, 1890.

DR. CHARLES K. MILLS in the Chair.

Dr. W. W. KEEN and Dr. F. X. DERCUM gave details of an operation, and the particulars of a case of spasmodic wry neck.

The following is an abstract of the remarks of Dr. Keen upon the operation.¹

A RESUME OF A NEW OPERATION FOR SPASMODIC WRY NECK, NAMELY, DIVISION OR EXSECTION OF THE NERVES SUPPLYING THE POSTERIOR ROTATOR MUSCLES OF THE HEAD.

By W. W. KEEN, M.D.,

Professor Principles of Surgery, Jefferson Medical College.

Three years ago, while studying carefully a case of spasmodic torticollis with Dr. S. Weir Mitchell, he asked me, in view of the implication of the posterior muscles of the neck which rotated the head, as well as of the sterno-cleido, whether it would be possible to do an operation for dividing or excising all the supplying nerves. A number of careful dissections determined the feasibility of the operation, and

¹ The full report will be published in "The Annals of Surgery," for January, 1891.

I have repeatedly done it on the cadaver, and once on the living subject. That case, Dr. Dercum, who kindly referred her to me at the Woman's Hospital for operation, will report fully to the Society this evening.

The chief posterior cervical muscles that rotate the head are the splenius capitis, the rectus capitis posticus major, and the obliquus inferior, of which the last, though not the largest muscle, has the most favorable leverage. The muscles are supplied by the posterior divisions of the first three cervical nerves.

[The anatomical relations of the parts involved were then pointed out, and especially their relations to the sub-occipital triangle formed by the rectus capitis posticus major and the two oblique muscles. The sub-occipital nerve emerges from this triangle, and the vertebral artery lies in it.

The steps of the operation were next described in detail, and it was pointed out that the hemorrhage, though free, was easily controllable, the drainage was perfect in the recumbent posture, and the recovery of the patient was speedy and uninterrupted.]

Those who desire to follow out the anatomy of the region, and the surgical steps attending it will find the details in the "Annals of Surgery" for January, 1891.

Dr. FRANCIS X. DERCUM.—The patient operated upon by Dr. Keen, was a woman, fifty-four years of age, who first presented herself at the University Hospital, March 27, 1886. She then stated that for two years past she had suffered from involuntary rotation of the head towards the left shoulder. As she spoke the movement frequently recurred. The chin turned toward the left and was slightly elevated. In addition, she had distinct hypertrophy of the sternocleido-mastoid muscle upon the right side. There was also some diffuse pain at the back of the neck, and at times dizziness, and occasionally headaches, but I could not determine that these were related to the spasm. She was operated upon June 2, 1888, by Dr. Ashhurst, at my suggestion, who removed four inches of the spinal accessory nerve, both branches being embraced in the operation and extreme traction being made upon the trunk. Following the operation the spasms disappeared for a week. They then returned and were apparently unchanged. The frequency was about the same as before. The patient then disappeared from view; but Dr. Keen, having communicated to me that he had devised a new operation for such cases, I wrote to her and placed her under Dr. Keen's care. Dr. Keen operated upon her, as he has described. I saw the

patient some time after the operation, and she told me that the spasm had at first entirely ceased, but at the end of one week the movement had returned, but that it was less violent than before. According to my observation, there was decided improvement, the rotation was not so marked, and the head was not moved so violently. She could steady the head with the hand, which was impossible before the operation.

No matter what operation is adopted in these cases, there is some return of the movement, indicating that we have a general condition to deal with, and which probably has its origin in some cortical disturbance. Many muscles are involved in producing this movement of rotation. In studying the subject some years ago, I found that nine different muscles were concerned in the rotation of the head, and at times eleven. It would therefore be impossible to cut all the nerves or to separate the insertions of all the muscles. In the case reported there was certainly improvement, and I think that if the patient had returned to have had an apparatus adjusted, the movement of the head could have been restrained almost entirely.

Dr. W. W. KEEN.—I saw the woman a year after the operation, and she could turn her head towards the left or right and retain it in that position for a considerable time. The trouble had returned, however, to some extent. The reason probably was that some of the nerves of the splenius had escaped division. All the other posterior muscles seemed to be free from contraction. This was the first case in which I performed this operation and I think it was not done as thoroughly as it might have been. It was, however, largely a success, as has been indicated by Dr. Dercum. The range of movement is lessened and its violence diminished; but it is not the complete success that I had hoped for. I propose the operation for trial and for an estimate of its worth. I cannot say that my own mind is made up in regard to this, although I think that it may give a certain amount of relief at least.

Dr. MORRIS J. LEWIS.—As Dr. Dercum has stated, we have many muscles to deal with in this condition. In studying this subject, I have found that according to "Gray's Anatomy," the muscles employed in rotating the head and turning the face to the *left* are situated upon both sides of the neck; upon the right side they are the sterno-cleido-mastoid, the trapezius, the obliquus superior, and the complexus; and on the left side, the splenius, the rectus capitis posticus major, the obliquus inferior, and the rectus anticus

major and minor. The case which I am about to report, shows the importance of the muscles upon the back of the neck in this rotation of the head, inasmuch as both sterno-cleido-mastoid muscles have been removed, together with the spinal accessory nerves, and yet the lateral movement continues.

SEVERE SPASM INVOLVING MUSCLES OF THE NECK, PERSISTING AFTER REPEATED SUR- GICAL PROCEDURES AND ACTIVE MEDICAL TREATMENT.

By MORRIS J. LEWIS, M D.

W. S., æt. 54, shoemaker and turner; denies syphilis. Present trouble began in 1878. Previous to this had been a healthy man. Used tobacco and alcohol in moderation. In 1865 fell on floor during a wrestling-match, his opponent falling on top of his head. In about one-half hour after this he had a good deal of pain in neck, but this soon entirely vanished.

He assigns no cause for his trouble: his first symptom was a tired aching feeling in back of neck and head, without marked twisting of head, except when he would attempt to look steadily at anything, and then his head would pull back and to the left. He does not remember the position of his chin at that time. His condition gradually got worse and worse, until in 1886 there was a continuous "pull of head to left," the chin being up in the air. At this time a seton was placed under the *left* trapezius and allowed to ulcerate through, which it took three weeks to do. This appeared to give temporary relief, but soon the condition was as bad as ever. Then a seton was placed under the sternal head of the *left* sterno-mastoid muscle; this was done two weeks after the former operation.

The position of the head was changed by these operations, but patient could not describe in what manner.

Then a seton was placed under the left splenius, and allowed to cut through. This produced no relief.

Finally a seton was placed under the lower end of right sterno-cleido-mastoid, but this was removed before it had separated. Thirteen weeks were occupied in these operations, and no relief was obtained.

After this, the tonic contraction of the muscles gave way to a clonic spasm of the head to the left, and the pain and discomfort became greater. In 1887, he began to take morphia, and has continued it ever since, sometimes in large doses. In 1887 he applied to the Orthopædic Hospital and Infirmary for relief. Rhigolene freezing of the skin over the right spinal accessory nerve was tried, with apparent good effect, all the symptoms, however, returning with full intensity in a short time.

In November, 1887, Dr. Mitchell admitted him into the hospital, and by his advice the *right* sterno-mastoid was excised and as much as possible of the right spinal accessory nerve removed. This relieved the clonic spasm, but the head still turned to the left as much as ever, the right ear, however, not being so much depressed.

Two weeks later, by some unaccountable mistake, the *left* sterno-mastoid was removed and the left spinal accessory excised. This operation produced no effect other than to weaken his power of turning the head to the *right*. Both the sterno-mastoids were removed in their entirety, not merely cut, and both spinal accessory nerves were removed as far as practicable during the operation. Yet, notwithstanding this, the patient's head turned strongly to the left, although he still possessed the power of turning the head past the middle line to the right.

He was again admitted to the hospital in June, 1890, and was under my care.

His head was turned strongly to left, the chin slightly elevated and four inches to left of median line, the *left* ear being one and three-quarter inches lower than *right*. The fibres of the *left* platysma were very prominent, and the right shoulder was decidedly drawn up.

No clonic spasm could be detected in the neck muscles, and there appeared to be considerable rigidity of the neck, as the head by force could scarcely be turned past the middle line to the right.

By voluntary effort patient could turn the chin one and one-half inches towards the median line, and about one inch further to the left than its usual position. He could elevate and depress the chin with considerable ease. The left omo-hyoid muscle was prominent and could easily be thrown into action. The edge of the left trapezius was tense, and the right still more so, and just anterior to the edge of the right trapezius the processes of the cervical vertebræ could be easily felt.

He complained of a dull, aching pain on the left side of the neck and over the left shoulder.

Neither arm could be raised above the head, although easily extended forward, the left arm being the weaker.

K. J. normal. E. J. present.

Dynamometer, R. 95; L. 95.

A fine tremor was detected in head for first time. He was still taking morphia.

His treatment consisted in weaning him from morphia, and placing him on increasing doses of gelsemium. Under this his general condition improved.

Cutting of the nerves on the back of the neck on the left side was advised, but to this the patient objected on account of the fear of paralysis and loss of control entirely of the head.

The muscles and nerves brought into action in rotating the head and turning the face to the left are, according to Gray and Flower, as follows:

NAME OF MUSCLE.	SIDE EMPLOYED.	NERVOUS SUPPLY.
Sterno-cleido-mastoid.	Right.	R. spinal accessory and deep branches of the cervical plexus.
Obliquus superior.	"	R. sub-occipital and great occipital.
Complexus.	"	R. cervical plexus and sub- and great-occipital.
Trapezius.	Left.	Left spinal accessory and deep branches of the cervical plexus.
Splenius capitis.	"	Left ext. post. branches of the cervical plexus.
Rectus cap. post. major.	"	Left sub- and great-occipital.
Obliquus inferior.	"	" " "
Rectus anticus major and minor.	"	Left sub-occipital and deep branches of cervical plexus.

Dr. F. X. DERCUM.—To the muscles which Dr. Lewis has mentioned, we should add those which are concerned in torsion of the cervical spine. The moment we admit the part which these muscles play, we see how impossible it is to absolutely limit the movement by operation.

SPASMODIC TORTICOLLIS UNSUCCESSFULLY TREATED BY LIGATURING THE SPINAL ACCESSORY NERVE WITH A SILVER WIRE.

By JOHN B. DEEVER, M.D., and CHARLES K. MILLS, M.D.

This interesting case has been much treated and much reported, and is brought to the Society with the hope that something further in the way of treatment may be suggested—something that will promise success. In a lecture

on "Spasmodic Torticollis" by Dr. Mills, in the "University Medical Magazine," February, 1890, and also in an article, entitled "Notes on the Action of Gelsemium in Local Spasm, etc.," by Dr. M. Imogene Bassette, in the "Journal of Nervous and Mental Disease," June, 1890, the case has been fully detailed. Both of these papers can be consulted for the record of the case prior to deciding upon the present operation. In brief, the case was one of a severe type of spasmodic torticollis, beginning some months after injury to the back of the head. She was treated with many remedies internally, and by repeated applications of the actual cautery at the back of the neck, but derived most benefit from frequently repeated and long-continued doses of gelsemium, as has been detailed in the paper of Dr. Bassette read before this Society. At one time it seemed as if a cure had been effected by means of this drug, but the patient was so thoroughly affected by it that it was thought unwise to continue it any further, and its use was finally abandoned. The spasm returned with almost all its old force and intensity. The sufferer, an unusually patient woman, still appealing to me for aid, and having read in the "Lancet" for June 21, 1890, an account of the successful treatment of a case by a silver wire ligature, I suggested to Dr. Deaver to try this operation.

The case in the "Lancet" is reported by Mayo Collier, M.S., Lond., F.R.C.S., Eng. The theory upon which he based his operation was, that as the disease consisted essentially of an excessive intermittent and irregular discharge of energy along the spinal accessory nerve, the introduction of increased resistance in the course of the nerve, and the possible deflection of some of this energy into other structures than the sterno-mastoid and trapezius muscles might result in an abatement or cure of the trouble. Collier's patient was a girl, twenty-one years old, and her affection dated from 1883. It was a most distressing case, the head being violently jerked every two seconds. She had been drugged, galvanized, blistered, and her tonsils removed. Collier cut down on the nerve, used some traction, and then placed silver wire around the nerve as high as he could reach, only twisting the ends to insure a slight compression. The skin was allowed to grow over the ends of the loop. The spasms entirely ceased. The operation was done in January of the present year, and on April 5th the patient wrote to the doctor that she was quite well.

DISCUSSION.

Dr. JOHN B. DEEVER.—In this case of Dr. Mills' I exposed the spinal accessory nerve at its point of exit from the sterno-cleido muscle, and ligatured it with silver wire, carried well up. The wound healed promptly. The loop of wire is encysted, and it occasions no inconvenience. In the light of Collier's result. This is a justifiable operation and worth repetition in this case, at least before adopting any other procedure.

I consider the operation devised by Dr. Keen a severe one, and not entirely justifiable procedure; not, at any rate, till other operative means have been tried. It certainly exposes a comparatively important part of the body, and, in the light of Collier's result, would it not be wise to try his (Collier's) operation, which does not involve any risk and does not leave the patient any worse?

Dr. W. W. KEEN.—I should like to make a few remarks in connection with this subject. A bad case presented itself at the Orthopædic Hospital on Saturday last. It was in a young child, and it was evident that several muscles were involved. The rotator muscles of the spine were affected, as well as the muscles of rotation of the head. Under the sterno-cleido muscle there was a mass of muscles which were tense and hard. These I took to be the scalene muscles, which aided in pulling the neck and head over to the right side. The levator anguli scapulæ was also involved. There was spasm of the sterno-cleido-mastoid muscle of one side, and of the posterior cervical muscles of the other side.

The whole question is a complicated one. Two years ago, when Mr. Horsley was in this country, I went over this matter with him, and we debated quite seriously the question of excision of the cortical centre for rotation of the head. I confess that I have never yet had the courage to do this operation, partly because I am in doubt whether it would reach so extensive a movement as that presented by Dr. Mills, where the muscles of both sides are affected, although very probably the function of this centre is bilateral. If a serious case presented itself to me, in which the annoyance and discomfort were of such a character that I thought it right to run any reasonable risk, I should recommend this excision of the cortex, especially if other operations had been done and failed, presuming of course that all medication had been tried. I think, however, that the position of the cortical centre of rotation of the head has not yet been ascertained with such accuracy that we can dogmatize.

Dr. JAMES HENDRIE LLOYD.—If Dr. Keen can, I wish he would tell us more definitely the position of this centre. In a recent lecture, Ferrier, referring to localization of the trunk muscles, finds their centres in the mesial aspect of the brain. He finds that excision of the centre of one side does not completely paralyze the trunk muscles. It seems to me that this would be an important point in connection with these cases. The muscles involved in torticollis might act as the trunk muscles do, and be paralyzed completely only by excision of the centres on both sides. Again, Ferrier says that lateral movements of the head and eyes have their centres in the frontal region and are not capable of being permanently paralyzed unless every portion of the frontal region be completely destroyed—a very formidable operation. In regard to its being a local central lesion in wry neck, that of course is still a matter to be decided; but I think all of us who have seen these obstinate cases of torticollis and have failed to obtain the slightest therapeutic result, and have seen all these extensive peripheral dissections made and peripheral treatment tried to the fullest extent, must feel that with such extensive associated movements as these, the chances are that we have not gone far enough back, and that the trouble is probably one located in the cortical centres.

Dr. W. W. KEEN.—In reply to Dr. Lloyd I would say that I have to-day received a copy of Ferrier's book, and I have not yet had time to read it. Mr. Horsley's view was that this centre lay on the lateral aspect of the hemisphere, and not on the mesial; and Ferrier's former view at least was the same. In the case reported by Dr. Mills I should be inclined to follow the suggestion of Dr. Deaver, to expose the nerve and again ligate it. I cannot exactly see the rationale of the operation; but as it has cured in one instance, it is worthy of a second trial.

It is scarcely just to refer to the operation I have described as a severe one; although it involves a number of muscles and nerves, yet the area is not nearly as large as that of many tumors which we remove without the slightest fear. There is not the least operative or post-operative risk, to my mind. If in Dr. Mills' case re-ligation of the nerve fails, I should advise the operation, which I have described, on the right side. I would rather do that than excise the cortical centre. If that did not succeed, it would be then a question whether the more radical operation should not be attempted.

Dr. G. BETTON MASSEY.—I know of nothing more obstinate than these inveterate cases of torticollis. If anything can be done surgically, it should be welcomed. I should advise more extensive exsections before concluding that the case was hopeless. Certainly the reports carry a certain grain of hope in them. I have looked at this affection as largely psychical, however, especially the clonic variety. One case that came under my observation some years ago seems to bear out that view. The spasm was much the same as in the case of Dr. Mills. This case resisted the most thorough treatment of all kinds, and finally went home and got well under the influence of exercise and fresh air.

FAILURE OF SCIATIC STRETCHING FOR A TYPICAL CASE OF SYMMETRICAL LATERAL SCLEROSIS.

BY DRs. W. W. KEEN AND CHARLES K. MILLS.

This patient, a young married woman, when she first came for treatment, had the symptoms of an incipient symmetrical lateral sclerosis, the disease being in such an early stage that some doubt existed as to whether we had not to deal with an hysterical rather than an organic spasmodic tabes. The sole symptoms were exaggerated kneejerks, slight ankle clonus, stiffness in the lower extremities, with a slightly halting and stumbling gait. Here was a good opportunity, if ever, of curing spinal sclerosis, and the patient was told that we would try to exhaust the measures known for its treatment. She proved a persevering patient, staying with Dr. Mills for several years, during which time she was treated with all manner of drugs, with galvanism, massage and the cautery, and for several months by forcibly stretching of the limbs without cutting down on the nerves. With the assistance of Dr. J. B. Roberts at one time she was etherized in order more thoroughly to carry out this method of sciatic stretching. In spite of all efforts she got slowly but surely worse, and finally Dr. Keen stretched both sciatics after incision.

The operation was performed on December 13, 1883, by Dr. Keen, the strictest antiseptic precautions being observed. The nerves were exposed by an incision just below the gluteal folds. A strong flat, curved hook was in-

roduced beneath each nerve, which was stretched first in one direction and then the other. The stretching was completed by suspending for a moment the entire leg by the hook placed under the nerve. Recovery from the operations was rapid and perfect. After the effect of the ether passed off, she had some numbness at the plantar surface of the left foot, most at the toes, and also slight loss of sensation along the outer edge of the sole of the foot. The plantar reflexes were exaggerated, but ankle clonus, which was present before the operation, had disappeared. The knee-jerks were still marked but less so than before the operation. Spontaneous tremors in the limbs were present at times, and she had occasionally pain in the left knee and ankle. These were the only points of interest in connection with the case after operation.

The woman remained for many months under observation. Her gait and reflexes seemed for a few weeks slightly better, but the improvement was neither marked nor permanent. The disease re-asserted itself, and steadily although very slowly got worse until she passed from observation.

TWO CASES OF SUCCESSFUL SCIATIC-STRETCHING.

BY DRS. CHARLES K. MILLS AND JOHN B. DEAVER.

The following cases are worthy of being reported, as in both the severe and long continued pain was relieved by operation :

Case 1.—This patient was an electrotyper, 32 years of age, without a history of syphilis, constitutional disease or abuse.

About three years before he came under observation, he was in the habit of frequently riding the bicycle, particularly in the evenings. Three months after beginning this amusement, which may, or may not, have had to do with his affection, he noticed pain in the under part of the left thigh. This persisted from day to day, and from week to week; it gradually increased in intensity. The pain seemed to start at a point about half way between the hip and the knee in the back of the thigh. It would extend in both directions from this point, so that the painful and uncomfortable sensation would reach the bottom of the foot, and in the other

direction the sacral region. At times he would have considerable numbness and tingling in the leg, particularly below the main focus of pain. He described the pain as being at times excruciating.

For three years before coming to the reporters he was treated by various physicians. Among other measures which were thoroughly tried, were electricity, both faradism and galvanism, the actual cautery, and many internal remedies, such as iodide, the salicylates, and antiperiodics, as arsenic, quinia, etc., also numerous anodynes. A course of massage and Swedish movements had also been tried, and he had been subjected to hypodermic injections.

He suffered mostly with this pain in the evening, and when sitting down during the day. When moving about the pain gradually decreased, and he was sometimes hardly aware of it, but it never absolutely left him.

We found that the pain was still most severe at the original spot about midway of the posterior thigh; that some numbness and tingling followed the posterior external aspect of the leg downward to the foot. Lately it seemed as if the pain had increased in the upper thigh and in the gluteal regions. On pressure no tenderness was present. We also made a careful examination by the rectum for the condition of the sacral plexuses, to determine whether or not a growth or any morbid condition was present in the pelvis. Nothing was discovered.

As the patient had already been treated by almost every measure promising success, except nerve stretching, after explaining this operation to him he consented to have it done.

With strict antiseptic precautions, Dr. Deaver exposed the nerve for several inches at the place of the greatest pain. No tumor could be detected. Some thickening and inflammation of the nerve sheath was present for about an inch and one-half or two inches. After opening the sheath and loosening the thickened bands, the nerve was carefully stretched by pulling it strongly first in one direction and then in the other.

It is unnecessary to go into the details of the subsequent condition of the patient. He suffered while in bed from various nervous symptoms the result of timidity and a nervous temperament. In about two weeks he was again on his feet. For several weeks he had the symptoms of traumatic neuritis in the sciatic distribution below the seat of the operation—spontaneous pain, and hyperæsthesia along the nerve distribution, and pain on lateral squeezing of the foot, but the old pain was gone.

After a few weeks of treatment with mercurial and belladonna ointments, salicylates, tonics, rest and hot applications, the neuritis which had resulted from the operation disappeared. He was also free from the old pain, and has remained so up to the time of making this report.

Case II.—T. J., aged 25, laborer, patient in the nervous wards of the Philadelphia Hospital, had been irregular in his habits, and was addicted to the abuse of alcohol, drinking, according to his way of putting it, "everything that came in his way." He never had had syphilis. In February, 1890, he complained of soreness of the hip and thigh. This soreness increased over the back of the leg and thigh, and in the upper portion of the foot; he also had a tingling and numbness in the lower part of the leg and foot. This was greatly increased by exertion or pressure. Lying in bed greatly relieved him, and, in consequence, for the sake of the comfort which he obtained, he often remained in bed two or three days at a time. In walking, semi-flexion of the leg apparently relieved the intense pain. Later this pain had extended to the gluteal regions.

For several months he was treated with the usual remedies which are employed in sciatic neuritis, including iodide, anti-periodics, salicylates and hypodermic injections of morphia and atropia. He improved somewhat, but no decided relief was obtained.

September 3, 1890, he was removed to the surgical wards of the hospital, where Dr. Deaver exposed and stretched both sciatic nerves. At first he had some of the neuritic discomfort which followed the other operation, but after a week he began to decidedly improve. In less than a month he was entirely free from pain in the leg and thigh.

SUTURE OF THE PERONEAL NERVE.

By JOHN B. DEAVER, M.D.

This is the only case of secondary suture of the peroneal nerve of which I can find any account. In Bowlby's book, if my memory serves me correctly, I find that secondary nerve suture has been practised a number of times in the upper extremity, and only once in the lower extremity.

The history of the case is as follows:

At the battle of Sharpsburg, 1862, J. R., aged twenty years, was struck over the trochanter major of right side

with an iron ball, penetrating to the bone; this was immediately followed by intense pain, causing patient to fall to the ground speechless. Upon rising and attempting to put right foot to the ground, he was again attacked with pain. Ten days later, when turning over on the left side to have the wound dressed, he suffered from pain, referred to outer side of knee of corresponding side. Four months from time of injury he resumed his duties in the army, being but little inconvenienced by the wound.

A year or two after the close of the war he noticed a small lump on outer side of right knee, which when struck caused pain, referred along the course of the branches of the external popliteal or peroneal nerve. The swelling slowly increased in sensitiveness and in size till it was about as large as a small English walnut. In April, 1890, the lump, which was evidently a neuroma (it was not examined microscopically), was removed by his family physician—no anæsthetic being used. The ends of the nerve were not sutured. This operation was followed by paralysis of the extensor muscles of the leg.

He wrote to a number of prominent surgeons; but they all disapproved of any operation. He came into my hands, when I thought it proper to make the attempt to suture the divided ends. I went to Virginia July 1st, and exposed the nerve, removing the two bulbar extremities, and sutured the ends with catgut and aseptic silk. On account of the amount of nerve removed, it was necessary to place the limb at an obtuse angle. The patient made an uninterrupted recovery, barring an attack of traumatic neuritis. As a result of this he became addicted to morphia.

Some months after the operation he came to Philadelphia, at my suggestion, and received electrical treatment with electricity, massage, and other measures under Dr. Mills. Sensation had returned. When he went back to Virginia the limb was almost straight, but there had been no recovery of motion.

There is no question but that the nerve has united. It can be distinctly felt, and pressure at the seat of suture causes pain along the musculo-cutaneous and anterior tibial branches. I believe that in the course of a year or two motion will be recovered. In the cases given by Bowlby, there are only one or two in which motion returned before the lapse of fifteen months.

I have here the two ends of the nerve. Bowlby states that the proximal end of the nerve is bulbous, and the distal end filamentary. Here both extremities are bulbous and united by fibrous tissue.

DISCUSSION.

Dr. CHARLES K. MILLS said that he treated this man for three or four weeks. He made a careful electrical examination, and found the reactions of degeneration. There was also active neuritis. This was treated by mercurial and belladonna ointment, freely applied, and the internal use of salicylates and tonics, with rest part of the time. Hot douches were also employed. This was followed by improvement. He examined the man frequently, and on one or two occasions thought that there were signs of return of motion in the muscles. His impression was that the man would eventually get back power in these muscles. The time that he was under treatment was not sufficient. Before the man returned to his home he was provided with an apparatus similar to that used in club-foot, the result of peroneal palsy; and with this he was able to get along pretty well.

Dr. W. W. KEEN thought that it would be important to have a microscopical examination of the bulbous extremities removed, to determine the presence or absence of nerve tissue. This should be done before any opinion was expressed upon the results of the operation.

One lesson that this case teaches distinctly is, that in all such cases the cut ends of the nerve should be at once united. This cannot be too strongly insisted upon. The profession should be educated up to this point. If the two ends had been united at the time of the removal of the neuroma, the chances of a useful limb would have been excellent.

TRIGEMINAL NEURALGIA OF LONG STANDING RELIEVED BY AN OPERATION.

REPORTED BY DRs. J. B. DEEVER AND CHARLES K. MILLS.

This patient was a man between seventy and eighty years of age. For many years he had been a carrier of newspapers, and constantly exposed to all kinds of weather. Several years before he came under care, he began to suffer with severe pain in the side of the face beneath and above the eye, and sometimes in front of the ear. The history of the case was the usual one of a gradually augmenting trigeminal neuralgia, or tic douloureux.

Many remedies were used by various physicians before he came to Dr. Mills. One of the branches of the fifth nerve had been resected by Dr. F. D. Gross. He was treated by Dr. Mills for several weeks, with galvanism, counter-irritants and internal medicines, including among others large doses of aconitia, which gave him temporary relief.

The patient was admitted to the Polyclinic Hospital and was operated upon by Dr. J. B. Roberts, for several months he was entirely free from pain. He then began to suffer again, and the attacks gradually increased both in severity and frequency, until soon they were practically continuous.

Applying again for treatment, he was admitted to the wards of Dr. Deaver, in the German Hospital.

Dr. JOHN B. DEAVER.—In this case of trigeminal neuralgia, the patient had been operated on twice—once by Dr. Roberts and once by Dr. Gross. My experience is that in these cases it is useless to do an operation on the terminal part of the nerve. I have operated on two cases particularly interesting. One of these was of two years standing and had been treated in Chicago, New York, and in this city. I removed the superior maxillary nerve with Meckel's ganglion. The operation was the ordinary Carnigan's operation. The man has been entirely relieved. The second patient, similarly treated, also has been well for two years.

HORSLEY'S NEW ROLANDIC FISSURE METER

was exhibited by Dr. W. W. Keen.

Dr. JOHN B. DEAVER.—What advantage has this instrument over the plan formerly used in locating this fissure? I have had occasion to make a number of observations in regard to the position of the fissure of Rolando with Dr. Agnew, and every time we found the fissure of Rolando without trouble. I think that the fewer instruments we have the better.

Dr. CHARLES K. MILLS.—What I think is probably of as much importance as this question of the angle of the fissure of Rolando is the position of the fissure in persons of different ages and different mental capabilities. A difference of almost, if not quite, an inch may be found according to the development of the brain with reference to age and mental capacity.

Dr. FRANCIS X. DERCUM.—I am in accord with Dr. Mills as regards the relation of the fissure of Rolando to the general brain development, but I also believe that there is

a distinct relation between the angle of the fissure of Rolando and the cranial index. From my own studies, I have no doubt that the angle is greater in brachycephalic, and smaller in dolichocephalic skulls.

Dr. W. W. KEEN.—I am not sufficiently informed to speak of the exact relation between the varying angle and the cranial index. If, as I believe, the fissure of Rolando does change its angle, it is of importance to have a rotatory arm, particularly in operations at the lower end of the fissure. A slight variation of one or two degrees may be of slight importance in the upper part of the fissure, but when we come to its lower extremity, such a variation will make a considerable difference. If we also neglect to take into account the genu of this fissure we may be one and a half or two centimetres out of our reckoning.

PROBABLE LESION OF THE POSTERIOR MEDIASTINUM.

By CHARLES K. MILLS, M.D.

This patient is of great interest in many ways; in one, as showing the method of evolution of a diagnosis.

J. E., aged forty-three, white, male, a park guard, was first seen on March 6, 1890. He had been in the hands of many physicians.

When he first appeared he was a robust-looking, well-developed man. His chief complaint was of severe pain in a spot about one to two inches below the inferior angle of the left shoulder-blade, with the arms at rest and pendant. At first the pain was scarcely noticeable, but later it became more persistent and its exacerbations more frequent. He was always worse in the afternoon and evening, apparently the result of exertion. The pain was increased to some extent by lifting a heavy weight, as a coal-scuttle or a bucket of water. As a peculiar manifestation, he complained that it hurt him to sneeze, unless he threw his head far back. When the pain was most severe it appeared to pass around and behind the ribs. Occasionally it reached to the middle line of the spine. After lying down for some time, he improved.

The pain was described as a dull, gnawing, grumbling sensation; but, as already stated, he at times had temporary decreases and exacerbations, some of the latter of a

darting character. He thought that talking, particularly if he spoke loudly, caused him to have a catch in the region of the pain. He sometimes had difficulty in getting his coat and other garments off and on.

The man was taken to the Polyclinic, and carefully examined by Dr. H. A. Wilson and the writer, with a view of determining whether he could have incipient caries; but the examination resulted negatively. I was inclined to the operation of cutting down upon the nerve and excising it, or perhaps tracing it back, in search of a lesion, to its origin in the vertebral canal.

Dr. Deaver saw the man, and arrangements were made for his admission to the German Hospital; but he preferred to wait, and to go on with medicinal treatment, counter-irritants, etc. He was treated by me for several weeks with no decided benefit. He then passed out of my hands.

Returning again to me about the middle of October—in the meantime having been treated by hypodermic injections, cauterization and in other ways—his condition was found to be much worse.

The most prominent symptom was still the grumbling, persistent pain below the shoulder-blade. The exacerbations were more violent, and the pain showed a tendency to radiate and diffuse. Occasionally he complained of pains leading to the spinal column behind. More frequently some pain passed upward between the left shoulder-blade and the vertebral column; again, at times, the pain passed up to the shoulder and down the arm.

He had now decidedly lost flesh, his weight having fallen twenty to thirty pounds. Several weeks before he came to me, an irritating cough had shown itself. This was not accompanied with much expectoration; occasionally it showed a little frothy mucus. He had frequent attacks of difficulty of breathing, even on moderate effort, and could not, without great dyspnoea, attempt any great exertion or violent effort. Heart palpitation occasionally occurred, and lately he had had frequent attacks of sweating, local in character and confined to the side of the head and neck. His expression was anxious, at least as compared with his former complaisant and healthful appearance. The left pupil was a little smaller than the right.

Being still somewhat uncertain of the nature of this painful disease, I brought him before the Philadelphia Neurological Society, submitting him to the members for examination and diagnosis.

[He was examined by nearly all the members present, among others by Drs. J. B. Deaver, T. J. Mays, James Tyson,

J. H. Lloyd, Morris Lewis, W. W. Keen, J. Madison Taylor, F. X. Dercum, J. P. C. Griffith, and G. B. Massey. Several suggestions were made, among them were aneurism of the thoracic aorta, neuroma, local neuritis, tuberculosis, and a probable small spinal unilateral growth, and local meningeal inflammation.]

Since the meeting the patient has again been examined by Dr. Mays and myself, and with him I have concluded that all the symptoms in the case are best explained by a lesion in the posterior mediastinum. The notes of Dr. Mays will be given at the conclusion of the report.

When the contents and relations of the posterior mediastinum are considered, it will be seen that, to explain the symptoms and progress of the affection, a lesion of the cavity would be the most reasonable diagnosis. Bounded in front by the pericardium and root of the lungs, behind by the spinal column, on either side by the pleura, the contents of this cavity are, in the first place, the two great nerve trunks, the pneumogastric and splanchnic nerves. Besides these, it contains the thoracic aorta, the azygos veins, the œsophagus, the thoracic duct and lymphatic glands. Irritation of the splanchnic and of the pneumogastric nerves with the recurrent laryngeal will explain such symptoms as change in the pupil, local sweating, etc. The persistent pain at its original seat, and the diffuse pain, is explicable on the theory of direct or indirect involvement of the spinal-thoracic nerves, which are connected with the splanchnics. Ross, of Manchester, has shown the connections of the sympathetic and the cerebro-spinal nerves with reference to the localization of pain, in lesions of the viscera, or the circum-visceral, or intra-visceral spaces.

If the location of the lesion be correct, the character of it is uncertain, as no aneurismal bruit has been heard. We may have a thoracic aneurism, a sarcoma, a lymphoma, tubercular disease of the nerves, or even vertebral caries, or vertebral cartilaginous or ligamentous disease. I believe it is possible to operate with a chance of success in a case of this kind, and I have suggested an operation to the patient.

After careful examination of this patient, Dr. Thomas J. Mays reported upon him as follows:

"Physical examination of this patient shows undue prominence of the upper part of the left chest, which is associated with a pulsating tumor, about one and a half inches square, located at the junction of the first intercostal space with the

sternum. Over this tumor there is marked dulness, which shades off toward the left and downward into a diminished percussion resonance. Over this dull area a diastolic murmur is heard, which is probably generated in the aortic valve. No systolic murmur anywhere. No asymmetry of the radial pulses, nor of the femoral and radial pulses.

"The respiratory sounds are partially suppressed in the whole of the left apex; whereas, in the right apex, there is puerile respiration. Some sibilant and mucous râles exist in the upper part of left lung.

"In coming to a conclusion concerning the nature of this trouble, I think it is evident that we have to deal with a growth or enlargement in the upper part of the chest and extraneous to the lung. There are no signs which indicate the existence of an aneurism, and I am of the opinion that there is a growth, probably lymphomatous or sarcomatous, in the posterior mediastinum pushing forward the arch of the aorta, and compressing the left bronchus, the left pneumogastric and the sympathetic chain on the same side. Whether the pain at the posterior base of the left chest is caused by this enlargement directly or in a reflex manner, I am not able to say, because there are no physical signs on the back to indicate whether the tumor extends down that far or not."

REMARKS ON TWO CASES SIMULATING HYDROPHOBIA AND OCCURRING AFTER DOG-BITES.

BY CHARLES K. MILLS, M.D., AND CHARLES J. HOBAN, M.D.

REMARKS BY DR. MILLS.

Owing to the grave importance of the subject to the comfort of society as well as its medical interest, it is well that cases alleged to be of hydrophobia in man should be placed for discussion before the profession. The weight of opinion is still in favor of the existence of a specific disease known as hydrophobia, both in the lower animals and in man, and the labors of Pasteur and his disciples have apparently given this affection bacteriological standing, but so carelessly and so recklessly are cases that will not bear

scrutiny reported as hydrophobia, that immense harm has been done both to the community and to the cause of science. In an article on "Cases with Hydrophobic Symptoms," published jointly with Drs. James Collins and Carl Seiler, in the "Philadelphia Medical Times," July 31, 1880, I directed attention to the fact that many of the cases reported as hydrophobia in man were really affections of a different character, the biting of a dog being merely a coincidence or the cause of a non-specific nerve affection like tetanus or reflex epilepsy. Among other diseases I then referred to as closely simulating hydrophobia were localized meningitis, tumors of the oblongata and floor of the fourth ventricle, epilepsy, tetanus and hysteria. This doctrine was not new with me. The elder Hammond had some years before contended that hydrophobia might be a nerve disease from the beginning, and the hypothesis is even as venerable as Democritus, who described hydrophobia as an inflammation of the nerves. During the last decade much interest has been awakened in the subject of pseudo-hydrophobia, and prominent in this work have been Dr. C. W. Dulles, of this city, and Dr. E. C. Spitzka, of New York, who seem inclined to take the advanced ground that hydrophobia is not a disease of man. Whether or not this view is correct, their work is deserving of commendation because of its tendency to counteract the tide towards nervous hydrophobia.

The first of the two following cases was seen by me in consultation with Drs. J. J. Healy, C. Wirgman and C. W. Dulles; the second with Dr. C. J. Hoban, who will report the case in full. In both were clear histories of dog-bites. Both were alleged to be cases of hydrophobia, but unfortunately a post-mortem examination could not be obtained in either. I do not believe that either was a case of genuine hydrophobia, for various reasons, but chiefly for two, namely: (1) While *some* of the symptoms in both corresponded to the description of hydrophobia in man, others were not at all of this character; (2) The symptoms in both cases could be much more readily explained by the supposition of a localized gross organic lesion. We have no right to conclude that a case is one of hydrophobia simply because the patient has a prior history of having been bitten at some period, near or remote, by a dog or other animal, but rather we should carefully exclude other possibilities before hazarding a conclusion so dangerous, directly and indirectly.

CASE I.—W. S., a young man, was seen in consultation December 27, 1880. His first symptoms had shown themselves three days before; but he had been bitten in the right hand several months previously. His first symptoms were general nervousness, irritability, apprehension, crying, and complaints of pain and weakness in his right arm. In a short time he lost power in the right arm. His chief symptoms, when he was examined jointly by Drs. Healy, Wirgman, Dulles and myself, were, in the first place, difficulty in swallowing water and other liquids. When, on urging, he attempted to drink, the liquid ran out of his mouth, because of paresis (not spasm) of some of the oral, facial and pharyngeal muscles. He had full control over the masseter, pterygoid and temporal muscles. The uvula was slightly deflected to the left and was congested, but it and the throat showed no deposit. The tongue was not paralyzed or paretic. The right upper extremity was partially and yet markedly paralyzed. His speech was slightly drawling and nasal, but he was not aphasic. He was a left-handed man. No loss of sensation was anywhere present; neither had he any hyperæsthesia. Temperature in right axilla 102.4° ; in left axilla, 102.4° . The heart action was fairly strong, without murmur, but with a roughness with second sound at the base. The right radial artery seemed smaller than the left, but no compensatory enlargement of the vessels of the other side could be determined. His tongue was heavily coated. He had no history of syphilis or traumatism. The patient died with symptoms of exhaustion and increasing respiratory difficulty within twenty-four hours after the consultation.

CASE SIMULATING HYDROPHOBIA AND OCCURRING SOME MONTHS AFTER A DOG-BITE.

By C. J. HOBAN, M.D.

H. S., male, age 5; head rather hydrocephaloid in appearance, showing no growth during last two years. When three month old had diphtheria, followed by whooping-cough. Both parents of nervous character, particularly father, who has had three attacks of mental trouble, during one of which he was under care of Dr. Mills. Had one attack about two years before birth of this child. Lost one

child one year old, a twin, under somewhat similar circumstances, having, however, no paralysis, dying in convulsions after an illness lasting twenty-four hours.

This child was bitten by a dog not supposed to be mad in the early part of March, present year. There were several lacerations, which bled slightly, on left hand. Wounds were washed with bichloride solution and carefully cauterized with aq. No. 3. Next day a few of the wounds were filled with pus, but in a few days were completely healed, leaving in time no scars whatever.

Was perfectly well up to October 3d, the beginning of his last illness. On October 3d at noon, the child complained of soreness in left wrist, the bitten hand; said that hand was tired. This tired feeling gradually extended up his arm, and by supper time there was complete paralysis of left arm, but no distinct pain was complained of. Parents applied arnica and iodine, but these gave no relief.

October 4th showed no change, nor did the following day. The boy kept running about as usual; went to Sunday-school and complained of nothing but loss of power in arm. On Sunday evening, October 5th, he complained of pain in left shoulder and became restless. On following morning, October 6th, I was sent for, and saw him for the first time about 1 P. M. Then there was complete paralysis of left arm, some pain, he said, in shoulder and neck. Was able to walk and run about, could eat and swallow as usual, but was somewhat nervous.

Pressure on arm, shoulder or neck gave no evidence of pain, no swelling was anywhere apparent. I looked upon it as a case of brachial paralysis, probably rheumatic in origin. Prescribed for it as such and said that he would recover the use of the arm in a few days. That evening I was sent for again about 7, and found that he had great difficulty in swallowing his medicine or any kind of liquid; there was also difficulty in swallowing solid food. Any attempt to swallow would be followed by spasmodic action of the larynx and œsophagus and part of the liquid would be thrown from the mouth again. At this time he was very nervous, and when I left he refused to make any attempt to swallow.

Saw him again on morning of October 6, and learned that he passed a very restless night, having no sleep, nor did he take any medicine or food. He was now most extremely nervous; the least noise or movement on the part of any one present would bring on marked convulsive

movements of the entire body, more marked in the upper extremities.

Pupils were widely dilated, pulse beating 180 times per minute and respiration very markedly increased. Mind was perfectly clear during all this, and the little patient would smile from time to time and declare that he had no pain.

On examination of his lungs, I found that they were rapidly becoming œdematous and respiration was getting difficult. Large quantities of saliva and mucus were secreted and expectorated in a foamy condition.

At 3 P. M., Dr. Mills saw him and found all these symptoms still more increased. In fact the child was so nervous now, that during examination he passed into such a convulsion that we expected him to die immediately.

His face became decidedly cyanosed, his lungs and trachea were filled with mucus so that it was barely possible for him to breath. However, on lowering his head and elevating his feet, he emptied his respiratory apparatus and again breathed with some difficulty.

He remained in this condition, having convulsive movements every few minutes until 7 P. M., when he died, perfectly conscious until within a few minutes of death.

The immediate cause of the death was cardiac and respiratory failure.

The cause of the paralysis of the arm and cardiac and respiratory failure is what I would like to learn to-night.

The evening the child died I thought I had a solution for all the symptoms, and wrote to Dr. Mills to that effect. In his answer he said my theory might be correct, but would be open to some criticism; and after giving the subject more attention, I certainly agree with him as far as the criticism is concerned. On looking over the plates in the fifth volume of Pepper's System of Practical Medicine, I located the brachial centre in the medulla oblongata, and putting this with the fact that the cardiac and respiratory centres are situated here and pressure on these would cause loss of function, I had an easy solution to all the symptoms. But on more reading, I find that the brachial centre is not so situated and of course my explanation as to the paralysis of the arm will not hold.

My theory was that there was a tumor situated near the medulla oblongata, which pressed first upon the brachial centre, causing the paralysis of the arm. This tumor by its rapid growth soon pressed upon the centre of the pneumogastric, causing the nerve to lose its inhibitory action over the heart; hence the rapidity of the pulse; pressure also on

the pneumogastric would cause the lungs to lose the power to throw off the mucus which accumulated so rapidly; hence the œdema.

But why tumor? In the first place the boy had no pain in head, nor any head symptoms whatever, and we can therefore exclude meningitis, cerebral effusions, and I think apoplexy. There was no cardiac trouble, and we may exclude embolism. Again, there was no pain in brachial plexus or arm, and hence we can exclude neuritis involving the plexus.

In the strict definition of the word, the child did not have hydrophobia, yet he had some of the symptoms of rabies, such as the foaming at the mouth, the pharyngeal and laryngeal spasms, the snapping at the towel when clearing his mouth of saliva, the peculiar noise made by attempts to rid his mouth of saliva, likened by one of the bystanders to the snarling of the dog. All these he had, but yet when we remember that there was paralysis of the left arm before any and all of the other symptoms, we cannot look upon it as a case of rabies.

Dr. JAMES HENDRIE LLOYD.—It seems to me a very violent assumption to consider that any of these clinical cases, either prove or disprove the existence of hydrophobia. If this disease exist it can be proved only by prolonged labors in the laboratory. I do not believe that newspaper reports or researches in libraries among musty books will settle the question. I suggest that when a physician gets a sensational case of so-called hydrophobia, he secure the cord and give it to a competent bacteriologist for study and experiment. Only in this way can we arrive at definite results. It is extremely improbable that fright produces death in the cases of so-called pseudo-hydrophobia. Fright is a constant phenomenon in human society, and its effects are very well understood. It would certainly be strange if it produces, as is asserted, such characteristic hydrophobic symptoms only after a dog-bite. Such a thing is not credible. It is probable, rather, that many reported cases have been badly observed, and have been cases really of delirium or maniacal excitement due to some infectious process in the brain.

Dr. G. BETTON MASSEY.—I have now a case presenting many of the symptoms detailed, referred to me by Dr. Chas. Martin of Allentown. Two years ago, when the acute attack occurred, the symptoms were much the same as those

of Dr. Hoban's case. There is now paresis of both arms without involvement of the nutritive condition, nor change in the normal response to electricity. At the beginning there was decided involvement of the throat and voice with a pulse at about 140. At present the pulse is 115 or 120. I consider the case to be one of lepto-meningitis of the upper part of the cord with a certain amount of pressure. If this individual had died early, it would probably have furnished food for the newspapers.

Dr. CHARLES K. MILLS.—I think that Dr. Lloyd's remarks are rather calculated to cloud the importance of these clinical reports of cases. I have reported several cases in which autopsies have been made. All the cases reported in the newspapers are sensational. Many of them come from France. It must be remembered that nineteen out of every twenty cases of so-called hydrophobia are undoubtedly not instances of this disease. There is a hydropho-hydrophobia which is the result of these reports. Both of these cases were spoken of by individuals as hydrophobia, and if for one moment assent had been given to this idea, the reports of the cases would have been sent broadcast throughout the country. These cases, as nearly all that I have seen, were clearly cases of something else.

Adjourned.

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A SUCCESSFUL CASE OF NEPHRECTOMY. By George Ben Johnston, M.D. Reprint.

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